ACTA·COLL·MED·PHILAD·INSTIT·A·D·MDCCLXXXVII·

Digitized by the Internet Archive
in 2013

http://archive.org/details/transactionsofco3151coll

TRANSACTIONS

OF THE

COLLEGE OF PHYSICIANS

OF

PHILADELPHIA.

THIRD SERIES.

VOLUME THE FIFTEENTH.

PHILADELPHIA:
PRINTED FOR THE COLLEGE.
1893.

NOTICE.

The present volume of Transactions contains the papers read before the College from January, 1893, to December, 1893, inclusive.

The Committee of Publication thinks it proper to say that the College holds itself in no way responsible for the statements, reasonings, or opinions set forth in the various papers published in its Transactions.

Note.—Owing to delays occurring in transmission, some of the papers contained in this volume are not placed in the order in which they were read.

DORNAN, PRINTER.

COLLEGE OF PHYSICIANS OF PHILADELPHIA.

1893.

OFFICERS AND STANDING COMMITTEES.

PRESIDENT.

S. WEIR MITCHELL, M.D.

VICE-PRESIDENT.

J. M. DA COSTA, M.D.

CENSORS.

William Goodell, M.D.,
Alfred Stillé, M.D.,
William Hunt, M.D.,
William F. Norris, M.D.

SECRETARY.

Charles W. Dulles, M.D.,

TREASURER.

Charles Stewart Wurts, M.D.

HONORARY LIBRARIAN.

Frederick P. Henry, M.D.

COUNCILLORS.

George C. Harlan, M.D.,
Caspar Morris, Jr., M.D.,
Hobart A. Hare, M.D.,
Henry R. Wharton, M.D.,
De Forest Willard, M.D.,
Thomas H. Fenton, M.D.

COMMITTEE OF PUBLICATION.

Arthur V. Meigs, M.D.,
Solomon Solis-Cohen, M.D. (to Feb. 1),
Robert P. Harris, M.D.,
G. G. Davis (from March 1).

LIBRARY COMMITTEE.

I. Minis Hays, M.D.,
Morris Longstreth, M.D.,
George C. Harlan, M.D.,
Barton C. Hirst, M.D.,
F. X. Dercum, M.D.
The Honorary Librarian *ex officio*.

COMMITTEE ON MÜTTER MUSEUM.

William Hunt, M.D.,
John H. Brinton, M.D.,
George McClellan, M.D.

HALL COMMITTEE.

J. Ewing Mears, M.D.,
William Barton Hopkins, M.D.,
Morris J. Lewis, M.D.,
Henry R. Wharton, M.D.,
J. K. Mitchell, M.D.

COMMITTEE ON DIRECTORY FOR NURSES.

William W. Keen, M.D.,
Wharton Sinkler, M.D.,
James C. Wilson, M.D.

COMMITTEE ON FINANCE.

John Ashhurst, Jr., M.D.,
Oliver A. Judson, M.D.,
William F. Norris, M.D.
The President and the Treasurer *ex-officio*.

COMMITTEE ON ENTERTAINMENTS.

Richard A. Cleemann, M.D.,
Louis Starr, M.D.,
J. Madison Taylor, M.D.,
John B. Shober, M.D.
The President *ex-officio*.

COMMITTEE ON WILLIAM F. JENKS PRIZE.

Horace Y. Evans, M.D.,
Edward P. Davis, M.D.,
Charles B. Penrose, M.D.

COMMITTEE ON ALVARENGA PRIZE.

J. William White, M.D.,
Alexander C. Abbott, M.D.,
J. Hendrie Lloyd,, M.D.
T. S. K. Morton, M.D.,
Frederick A. Packard, M.D.

TRUSTEES OF WILLIAM J. JENKS MEMORIAL FUND.

James V. Ingham, M.D. (orig.), Wm. S. W. Ruschenberger, M.D. (orig.),
Charles Stewart Wurts, M.D. (elected by Council March 26, 1890).

LIST

OF THE

PRESIDENTS OF THE COLLEGE FROM THE TIME OF ITS INSTITUTION.

ELECTED

1787. JOHN REDMAN.
1805. WILLIAM SHIPPEN.
1809. ADAM KUHN.
1818. THOMAS PARKE.
1835. THOMAS C. JAMES.
1835. THOMAS T. HEWSON.
1848. GEORGE B. WOOD.
1879. W. S. W. RUSCHENBERGER.
1883. ALFRED STILLÉ.
1884. SAMUEL LEWIS.
1884. J. M. DA COSTA.
1886. S. WEIR MITCHELL.
1889. D. HAYES AGNEW.
1892. S. WEIR MITCHELL.

FELLOWS

OF THE

COLLEGE OF PHYSICIANS OF PHILADELPHIA.

DECEMBER, 1893.

* Non-resident Fellows.
† Fellows who have commuted dues.

ELECTED

1883. Abbot, Griffith E., Ph.D., M.D.

1892. Abbott, A. C., M.D., First Assistant, Laboratory of Hygiene, University of Pennsylvania.

1870. Adler, John M., M.D.

1876. Alison, Robert H., M.D.

1867. Allen, Harrison, M.D., Professor of Zoölogy and Comparative Anatomy in the University of Pennsylvania.

1873. Allis, Oscar H., M.D., Clinical Lecturer on Orthopædic Surgery in Jefferson Medical College, and Surgeon to the Hospital of the same; Surgeon to the Presbyterian Hospital.

1888. Anders, James M., M.D., Professor of Hygiene and Clinical Diseases of Children in the Medico-Chirurgical College, Philadelphia; Physician to the Philadelphia Hospital.

1869. Andrews, T. Hollingsworth, M.D., Consulting Surgeon to the Hospital of the Good Shepherd, Radnor; Medical Director of the Bureaus of Police and Fire of the Department of Public Safety.

*1882. Ashbridge, Richard, M.D., Assistant Surgeon U. S. Navy.

ELECTED

1863. Ashhurst, John, Jr., M.D., Professor of Surgery in the University of Pennsylvania; Surgeon to the Pennsylvania and the Children's Hospitals; Consulting Surgeon to St. Christopher's and the Woman's Hospitals, and to the Hospital of the Good Shepherd, Radnor.

1865. Ashhurst, Samuel, M.D., Surgeon to the Children's Hospital.

1893. Ashton, Thomas G., M.D.

1857. Atlee, Walter Franklin, A.M., M.D., Corresponding Member of La Société des Sciences Médicales de Lyons; Consulting Surgeon to St. Luke's Hospital, Bethlehem; Visiting Physician to the Preston Retreat.

*1852. Bache, Thomas Hewson, M.D., Rome, Italy.

1883. Baer, Benjamin F., M.D., Professor of Gynecology in the Philadelphia Polyclinic.

†1892. Baker, George Fales, B.S., M.D., Surgeon to Outpatient Department of St. Joseph's Hospital.

1879. Baker, Washington H., M.D., Obstetrician to the Maternity Hospital.

1889. Baldy, John Montgomery, M.D., Professor of Gynecology in the Philadelphia Polyclinic; Surgeon to Gynecean Hospital; Gynecologist to Pennsylvania Hospital.

1880. Bartholow, Roberts, M.D., Professor (Emeritus) of Materica Medica, General Therapeutics, and Hygiene in Jefferson Medical College.

1883. Baum, Charles, M.D., A.M., Ph.D.

1873. Baxter, H. F., M.D.

1883. Beates, Henry, M.D.

1860. Benner, Henry D., M.D.

1874. Bennett, W. H., M.D., Physician to St. Christopher's Hospital for Children, and to Children's Seashore House, Atlantic City.

†1884. Biddle, Alexander W., M.D.

1884. Biddle, Thomas, M.D.

*1866. Black, J. J., M.D., New Castle, Del.

ELECTED

*1867. Boardman, Charles H., M.D., Boston, Mass.

1859. Boker, Charles S., M.D.

1891. Boyd, George M., M.D., Physician to the Lying-in Charity; Surgeon to the Out-door Department Episcopal Hospital; Assistant Surgeon to the Kensington Hospital for Women.

†1884. Bradford, Thomas Hewson, M.D., Physician to the Dispensary of the Children's Hospital and to the Gynecological Departments of the Pennsylvania and the Howard Hospitals.

1856. Brinton, John H., M.D., Professor of the Practice of Surgery and of Clinical Surgery in the Jefferson Medical College; Consulting Surgeon to the Southwestern Hospital of Philadelphia.

1891. Brinton, Lewis, M.D., Visiting Physician to the Nervous Department of Howard Hospital.

1887. Brubaker, Albert P., M.D., Professor of Physiology and General Pathology in the Pennsylvania College of Dental Surgery; Demonstrator of Physiology in Jefferson Medical College; Lecturer on Anatomy and Physiology at the Drexel Institute.

*1890. Brush, Edward N., M.D., Medical Superintendent of the Sheppard Asylum, Towson, Md.

*1851. Bullock, William R., M.D., Wilmington, Del.

1887. Bunting, Ross R., M.D.

1870. Burnett, Charles H., M.D., Clinical Professor of Otology in the Woman's Medical College; Professor (Emeritus) of Otology in the Philadelphia Polyclinic.

1892. Burr, Charles W., M.D., Visiting Physician to St. Joseph's Hospital, and the Home for Incurables; Visiting Pathologist to the State Asylum for the Insane.

1886. Cadwalader, Charles E., M.D.

1892. Cattell, Henry W., M.D., Demonstrator of Morbid Anatomy in the University of Pennsylvania; Pathologist to the Presbyterian Hospital.

ELECTED

*1892. Cerna, David, M.D., Ph.D., Demonstrator of Physiology in the Department of Medicine of the University of Texas; Corresponding Fellow of the Sociedad Española de Higiene of Madrid; Galveston, Texas.

1885. Chapin, John B., M.D., Physician to the Pennsylvania Hospital for the Insane.

1880. Chapman, Henry C., M.D., Professor of the Institutes of Medicine and of Medical Jurisprudence in Jefferson Medical College.

1868. Cheston, D. Murray, M.D.

1873. Clark, Leonardo S., M.D.

1872. Cleeman, Richard A., M.D.

*1842. Clymer, Meredith, M.D., New York.

1871. Cohen, J. Solis, M.D., Professor (Emeritus) of Diseases of the Throat and Chest in the Philadelphia Polyclinic; Professor (Honorary) of Laryngology in Jefferson Medical College; Consulting Physician to the Home for Consumptives, Philadelphia.

1888. Cohen, Solomon Solis, M.D., Professor of Clinical Medicine and Applied Therapeutics in the Philadelphia Polyclinic, and Physician to the Polyclinic Hospital; Clinical Lecturer on Medicine in Jefferson Medical College; Visiting Physician to the Philadelphia Hospital; Consulting Physician to the Jewish Hospital.

1866. Cruice, R. B., M.D., Surgeon to St. Joseph's Hospital.

1884. Curtin, R. G., M.D., Lecturer on Physical Diagnosis in the University of Pennsylvania; Assistant Physician to the University Hospital; Physician to the Philadelphia and Presbyterian Hospitals.

1884. Da Costa, John C., M.D., Gynecologist to Jefferson Medical College Hospital and to St. Agnes' Hospital.

†1858. Da Costa, J. M., M.D., LL.D., Professor (Emeritus) of the Principles and Practice of Medicine in Jefferson Medical College; Physician to the Pennsylvania Hospital; Consulting Physician to the Children's Hospital and to the Northern Dispensary.

ELECTED

1887. Daland, Judson, M.D., Instructor in Clinical Medicine in the University of Pennsylvania; Assistant Physician to the University Hospital; Physician to the Rush Hospital for Consumption.

1859. Darrach, James, M.D., Consulting Surgeon to the Germantown Hospital.

1888. Davis, Edward P., M.D., Professor of Obstetrics and Diseases of Children in the Philadelphia Polyclinic; Demonstrator of Obstetrics in Jefferson Medical College; Visiting Obstetrician to the Philadelphia Hospital.

1889. Davis, G. G., M.D., M.R.C.S. Eng., Surgeon to the German and St. Joseph's Hospitals; Assistant Surgeon to the Orthopædic Hospital; Assistant Demonstrator of Surgery in the University of Pennsylvania.

1874. Deakyne, A. C., M.D.

1887. Deaver, John B., M.D., Associate Professor of Anatomy in the University of Pennsylvania; Surgeon to the Philadelphia Hospital, to the German Hospital, and to St. Mary's Hospital.

1892. Deaver, Richard Wilmot, M.D.

1885. Dercum, Francis X., M.D., Clinical Professor of Neurology in Jefferson Medical College; Neurologist to the Philadelphia Hospital.

1891. Dixon, Samuel G., M.D., Professor of Microscopic Technology and Histology, and Curator of the Academy of Natural Sciences of Philadelphia.

1891. Dixon, William C., M.D., Physician to Industrial Home for Blind Women, Philadelphia; Physician to the Shelter for Colored Orphans, Philadelphia; Member of Consulting Staff, Philadelphia Home for Incurables; Examiner of Insane Patients, Philadelphia Hospital.

1893. Downs, Norton, M.D.

1884. Downs, R. N., M.D.

1884. Drysdale, T. M., M.D.

1864. Duer, Edward L., M.D., Accoucheur to the Philadelphia Hospital; Surgeon to the Maternity Hospital; Visiting Physician to the Preston Retreat.

ELECTED

1871. Duhring, L. A., M.D., Professor of Skin Diseases in the University of Pennsylvania.

1881. Dulles, Charles Winslow, M.D., Surgeon to Rush Hospital; Surgeon to Out-patients, Presbyterian Hospital; Lecturer on History of Medicine, University of Penna.

1863. Dunglison, Richard J., M.D.

*1871. Dunglison, Thomas R., M.D., Paris, France.

1888. Dunn, Thomas D., M.D.

*1849. Dunnott, Justice, M.D., Harrisburg, Pa.

1860. Dunton, William R., M.D., Consulting Physician to the Germantown Hospital.

*1882. Edwards, Joseph F., M.D., Atlantic City, N. J.

*1887. Edwards, William A., M.D., San Diego, California.

1893. Eshner, Augustus A., M.D., Adjunct Professor of Clinical Medicine in the Philadelphia Polyclinic; Registrar in the Neurological Department of the Philadelphia Hospital.

*1880. Eskridge, J. T., M.D., Denver, Colorado.

1868. Evans, Horace Y., M.D, Physician to the Charity Hospital.

1893. Farr, William W., Assistant Surgeon to the Genito-urinary Dispensary of the Philadelphia Polyclinic.

1884. Fenton, Thomas H., M.D.

1866. Fischer, Emil, M.D.

1884. Fisher, Henry M., M.D., Physician to the Episcopal Hospital; Microscopist to the Pennsylvania Hospital, and Physician to the Out-patient Department of the same.

1888. Flick, Lawrence F., M.D.

1862. Forbes, William S., M.D., Professor of Anatomy in Jefferson Medical College.

1870. Ford, William H., M.D., President of the Board of Health of Philadelphia; Physician to the Foster Home.

†1885. Fox, Joseph M., M.D., Leesburg, Va.

1890. Freeman, Walter, J., M.D., Adjunct Professor of Laryngology in the Philadelphia Polyclinic; Laryn-

ELECTED

gologist to the Out-patient Department of the Children's Hospital.

1885. Fricke, Albert, M.D.

1893. Friebis, George, M.D., Ophthalmic Surgeon to the Mary Drexel Home and to the German Hospital.

1889. Fussell, M. Howard, M.D., Chief Physician to the Medical Dispensary of the University of Pennsylvania; Instructor of Clinical Medicine in the University of Pennsylvania.

1873. Gerhard, George S., M.D.

1884. Getchell, F. H., M.D.

1892. Gibb, Joseph S., M.D., Instructor in Diseases of Throat and Nose in the Philadelphia Polyclinic; Surgeon to the Ear, Nose, and Throat Department of the Episcopal Hospital.

1885. Girvin, Robert M., M.D., Gynecologist to the Presbyterian Hospital.

1889. Githens, William H. H., M.D., Visiting Physician to The Sheltering Arms.

*1893. Gobrecht, William, M.D., Washington, D. C.

1884. Godey, Harry, M.D.

1868. Goodell, William, M.D., Honorary Professor of Clinical Gynecology in the University of Pennsylvania; Consulting Physician to the Lying-in Department of the Northern Dispensary.

1893. Goodell, W. Constantine, M.D., Clinical Instructor of Gynecology in the University of Pennsylvania; Assistant Gynecologist.

1867. Goodman, H. Ernest, M.D., Professor of Surgery in the Medico-Chirurgical College; Surgeon to Wills Eye Hospital and to the Orthopædic Hospital; Consulting Surgeon to the Maternity Hospital.

1885. Graham, John, M.D.

1891. Green, Walter D., A.M., M.D., Out-patient Surgeon to the Pennsylvania, Children's, and Methodist Hospitals; Assistant Surgeon to the Gynecean Hospital.

ELECTED

1870. Grier, M. J., M.D.

1883. Griffith, J. P. Crozer, M.D., Clinical Professor of the Diseases of Children in the Hospital of the University of Pennsylvania; Professor of Clinical Medicine in the Philadelphia Polyclinic; Physician to St. Agnes', the Children's, the Howard, and Rush Hospitals.

1871. Grove, John H., M.D., Surgeon to St. Mary's and to St. Agnes' Hospitals

1889. Guitéras, John, M.D., Professor of General Pathology and Morbid Anatomy in the University of Pennsylvania.

1863. Hall, A. Douglass, M.D., Surgeon to Wills Eye Hospital. (Resigned January 3, 1894.)

1893. Hamill, Robert H., M.D.

*1859. Hammond, William A., M.D., Surgeon-General U.S.A. (Retired), Washington, D. C.

1886. Hansell, Howard F., M.D., Chief Clinical Assistant to the Ophthalmological Department of Jefferson Medical College Hospital; Ophthalmic and Aural Surgeon to the Southwestern Hospital.

1889. Hare, Hobart A., M.D., Professor of Therapeutics in Jefferson Medical College; Physician to St. Agnes' Hospital and to Jefferson Medical College Hospital.

1865. Harlan, George C., M.D., Surgeon to Wills Eye Hospital and to the Eye and Ear Department of the Pennsylvania Hospital; Professor (Emeritus) of Diseases of the Eye in the Philadelphia Polyclinic.

1863. Harlow, Lewis D., M.D.

1862. Harris, Robert P., M.D.

1885. Harte, Richard H., M.D., Demonstrator of Osteology in the University of Pennsylvania and Assistant Surgeon to the Hospital; Surgeon to the Pennsylvania Hospital, to the Episcopal Hospital, and to St. Mary's Hospital; Consultant Surgeon to St. Timothy's Hospital.

1851. Hartshorne, Henry, M.D., LL.D.

ELECTED

1888. Hartzell, Milton B., M.D., Assistant Physician to the Dispensary for Skin Diseases, University of Pennsylvania.

1872. Hays, I. Minis, M.D.

1882. Hearn, W. Joseph, M.D., Surgeon to the Hospital of Jefferson Medical College and to the Philadelphia Hospital.

1884. Henry, Frederick P., M.D., Physician to Jefferson Medical College Hospital and to the Philadelphia Hospital; Professor of the Principles and Practice of Medicine in the Woman's Medical College of Pennsylvania.

1891. Hewson, Addinell, A.M., M.D., Demonstrator of Anatomy in Jefferson Medical College; Chief Clinical Assistant in the Surgical Dispensary of Jefferson Medical College Hospital; Dispensary Surgeon to the Hospital of the Protestant Episcopal Church.

1872. Hinkle, A. G. B., M.D.

1892. Hinsdale, Guy, M.D., Lecturer on Climatology in the University of Pennsylvania; Physician to the Presbyterian Orphanage and to the Out-patient Department of the Presbyterian Hospital; Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Curator of the Mütter Museum.

1888. Hirsh, Abram B., M.D.

1888. Hirst, Barton Cooke, M.D., Professor of Obstetrics in University of Pennsylvania; Obstetrician to the Philadelphia Hospital and to the Maternity Hospital.

1885. Holland, James W., M.D., Professor of Medical Chemistry and Toxicology in Jefferson Medical College.

†1879. Hopkins, William Barton, M.D., Surgeon to the Episcopal Hospital and to the Out-patient Department of the Pennsylvania Hospital.

1867. Horn, George H., M.D., Professor of Entomology in the Biological Department of the University of Pennsylvania.

1888. Horwitz, Orville, M.D., Clinical Professor of Genito-urinary Diseases in Jefferson Medical College; Surgeon to the Philadelphia Hospital.

ELECTED

1868. Howell, Samuel B., M.D., Professor of Chemistry in the Medico-Chirurgical College.

1892. Hughes, Wm. E., M.D.

1854. Hunt, William, M.D., Surgeon to the Pennsylvania Hospital.

1871. Ingham, James V., M.D.

1885. Jackson, Edward, M.D., Professor of Diseases of the Eye in the Philadelphia Polyclinic; Surgeon to Wills Eye Hospital; Ophthalmologist to Rush Hospital.

1887. Jayne, Horace, M.D., Professor of Vertebrate Morphology in the Biological Department of the University of Pennsylvania.

1885. Judd, Leonardo Da Vinci, M.D.

1867. Judson, Oliver A., M.D.

1886. Jurist, Louis, M.D., Chief Clinical Assistant in the Laryngological Department of Jefferson Medical College Hospital.

1849. Keating, William V., M.D., Physician to St. Joseph's Hospital.

†1867. Keen, William W., M.D., LL.D., Professor of the Principles of Surgery and of Clinical Surgery in Jefferson Medical College; Surgeon to Jefferson Medical College Hospital, and to the Orthopædic Hospital and Infirmary for Nervous Diseases, and Consulting Surgeon to St. Agnes' Hospital and to the Woman's Hospital.

*1887. Kelly, Howard A., M.D., Professor of Gynecology in the Johns Hopkins University, and Gynecologist and Obstetrician to the Hospital, Baltimore, Md.

*1844. King, Charles R., M.D., Andalusia, Pa.

†1875. Kirkbride, Joseph J., M.D.

1892. Lainé, Damaso T., M.D.

*1865. La Roche, C. Percy, M.D., Rome, Italy.

ELECTED

1887. Leaman, Henry, M.D.

1893. Le Conte, Robert G., M.D., Surgeon to Out-patient Department of the Pennsylvania Hospital, Children's Hospital, and Methodist Episcopal Hospital; Assistant Surgeon to the Gynecean Hospital; Assistant Surgeon to the Genito-urinary Department of the University of Pennsylvania; Assistant Surgeon, 3d Regiment N. G. of Pa.

1883. Leffmann, Henry, M.D., Professor of Chemistry in the Philadelphia Polyclinic and in the Woman's Medical College; Pathological Chemist to Jefferson Medical College Hospital.

1892. Leidy, Joseph, M.D., Physician to Out-patient Department of the Pennsylvania Hospital; Assistant Surgeon to the Genito-urinary Department of the University of Pennsylvania.

1855. Lewis, Francis W., M.D.

1877. Lewis, Morris J., M.D., Physician to the Children's Hospital, to the Orthopædic Hospital and Infirmary for Nervous Diseases, and to the Pennsylvania Hospital.

1886. Lloyd, J. Hendrie, M.D., Physician to the Nervous and Insane Department of the Philadelphia Hospital, to the Methodist Episcopal Hospital, and to the Home for Crippled Children.

1893. Longaker, Daniel, M.D.

1877. Longstreth, Morris, M.D., Professor of Pathological Anatomy in Jefferson Medical College; Physician to the Pennsylvania Hospital.

1886. MacCoy, Alexander W., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic; Lecturer on Diseases of the Throat and Nose in the Woman's Medical College of Pennsylvania.

1875. McClellan, George, M.D., Surgeon to the Howard Hospital.

1871. McFerran, J. A., M.D.

*1885. Mallet, John W., M.D., Charlottesville, Va.

ELECTED

1893. Marshall, John, M.D., Assistant Professor of Chemistry in the University of Pennsylvania.

1889. Martin, Edward, M.D., Surgeon to the Howard Hospital; Clinical Professor of Genito-urinary Surgery to the Hospital of the University of Pennsylvania.

1887. Massey, Isaac, M.D., Surgeon to the Pennsylvania Railroad; Physician to the Chester County Hospital.

*1850. Mayer, Edward R., M.D., Wilkesbarre, Pa.

1885. Mays, Thomas J., M.D., Professor of Diseases of the Chest and of Experimental Therapeutics in the Philadelphia Polyclinic; Visiting Physician to Rush Hospital.

1868. Mears, J. Ewing, M.D., Professor of Anatomy and Clinical Surgery in the Pennsylvania College of Dental Surgery; Gynecologist to Jefferson Medical College Hospital; Surgeon to St. Agnes' Hospital.

1875. Meigs, Arthur V., M.D., Physician to the Pennsylvania Hospital; Consulting Physician to the Pennsylvania Institution for the Instruction of the Blind.

*1884. Mifflin, Houston, M.D., Columbia, Pa.

1881. Mills, Charles K., M.D., Professor of Diseases of the Mind and Nervous System in the Philadelphia Polyclinic; Clinical Professor of Mental Diseases in the University of Pennsylvania, and of Nervous Diseases in the Woman's Medical College; Neurologist to the Philadelphia Hospital, and Consulting Physician to the Department for the Insane of the Philadelphia Hospital.

†1888. Mitchell, John K., M.D., Instructor in Clinical Medicine in the University of Pennsylvania; Physician to St. Agnes' Hospital; Assistant Physician to the University Hospital and to the Infirmary for Nervous Diseases.

1856. Mitchell, S. Weir, M.D., Professor of Diseases of the Mind and Nervous System in the Philadelphia Polyclinic; Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Consulting Physician to the Maternity Hospital.

1882. Montgomery, Edward E., M.D., Clinical Professor of

ELECTED

Gynecology in the Jefferson Medical College; Obstetrician to the Philadelphia Hospital.

1863. Morehouse, George R., M.D., Ph.D.

1886. Morris, Caspar, M.D., Physician to the Episcopal Hospital and to the Out-patient Department of the Pennsylvania Hospital.

1893. Morris, Elliston J., M.D.

1883. Morris, Henry, M.D., Gynecologist to the Howard Hospital.

1856. Morris, J. Cheston, M.D.

1861. Morton, Thomas G., M.D., Surgeon to the Pennsylvania and the Orthopædic Hospitals; Consulting Surgeon to the Jewish Hospital; Emeritus Surgeon to Wills Eye Hospital.

1891. Morton, T. S. K., M.D., Professor of Surgery in the Philadelphia Polyclinic; Surgeon to the Polyclinic Hospital; Assistant Surgeon to the Orthopædic Hospital; Surgeon to Out-patient Department of the Pennsylvania Hospital.

1864. Moss, William, M.D.

1890. Müller, Auguste F., M.D., Attending Physician to the Germantown Hospital.

1882. Musser, John H., M.D., Assistant Professor of Clinical Medicine in the University of Pennsylvania; Physician to the Philadelphia Hospital and to the Presbyterian Hospital; Consulting Physician to the Woman's Hospital of Philadelphia, and to the West Philadelphia Hospital for Women.

1886. Neff, Joseph F., M.D.

1887. Neilson, Thomas Rundle, M.D., Surgeon to the Episcopal Hospital and to St. Christopher's Hospital for Children; Professor of Genito-urinary Diseases in the Philadelphia Polyclinic; Lecturer on Diseases of the Rectum, and Assistant Demonstrator of Anatomy in the University of Pennsylvania.

1889. Noble, Charles P., M.D., Surgeon-in-Chief to the Ken-

ELECTED

sington Hospital for Women; Surgeon-in-Charge of the Department for Women of the Northern Dispensary; Surgeon-in-charge of the Department for Women of the Union Dispensary; Lecturer on Gynecology in the Philadelphia Polyclinic.

1893. Noble, William H., M.D.

1869. Norris, Herbert, M.D., Supervising Physician to St. Clement's Hospital.

1865. Norris, Isaac, Jr., M.D.

1892. Norris, Richard C., M.D., Demonstrator of Obstetrics, University of Pennsylvania; Assistant Obstetrician, University Maternity; Obstetric Registrar, Philadelphia Hospital; Visiting Physician to the Methodist Hospital; Consulting Obstetrician and Attending Gynecologist to the Southeastern Dispensary.

1866. Norris, William F., M.D., Honorary Professor of Ophthalmology and Clinical Professor of Diseases of the Eye in the University of Pennsylvania; Surgeon to Wills Eye Hospital.

1884. Oliver, Charles A., M.D., Attending Surgeon to Wills Eye Hospital; Ophthalmic Surgeon to the Presbyterian Hospital; Consulting Ophthalmic Surgeon to St. Agnes', St. Timothy's, and the Maternity Hospitals.

1884. O'Neill, J. W., M.D.

*1885. Osler, William, M.D., Professor of Medicine in Johns Hopkins University, and Physician to the Hospital, Baltimore, M.D.

1890. Packard, Frederick A., M.D., Visiting Physician to the Episcopal and Methodist Hospitals; Physician to the Out-patient Department of the Pennsylvania and Children's Hospitals; Instructor in Physical Diagnosis in the University of Pennsylvania.

1858. Packard, John H., M.D., Surgeon to the Pennsylvania Hospital and to St. Joseph's Hospital.

1864. Pancoast, William H., M.D., Professor of Anatomy

ELECTED

and of Clinical Surgery in the Medico-Chirurgical College; Consulting Surgeon to the Philadelphia Hospital for Skin Disease.

1882. Parish, William H., M.D., Professor of Obstetrics in the Dartmouth Medical College; Professor of Anatomy in the Woman's Medical College of Pennsylvania; Consulting Obstetrician to the Lying-in Charity; Consulting Surgeon to the Kensington Hospital; Consulting Gynecologist to St. Agnes' Hospital.

1883. Parvin, Theophilus, M.D., Professor of Obstetrics and Diseases of Women and Children in the Jefferson Medical College.

†1889. Penrose, Charles Bingham, M.D., Clinical Professor of Gynecology in the University of Pennsylvania; Surgeon to the Gynecean Hospital.

1854. Penrose, R. A. F., M.D., LL.D., Professor (Emeritus) of Obstetrics and Diseases of Women and Children in the University of Pennsylvania; Consulting Obstetrician to the Maternity Hospital; Visiting Physician to the Preston Retreat.

1868. Pepper, William, M.D., LL.D., Provost of the University of Pennsylvania, and Professor of the Theory and Practice of Medicine in the same.

1884. Perkins, Francis M., M.D., Ophthalmic Surgeon to St. Agnes' Hospital; Ophthalmic Surgeon to the Presbyterian Hospital.

1890. Phillips, J. Willoughby, M.D.

1883. Piersol, George A., M.D., Professor of Anatomy in the University of Pennsylvania.

1872. Porter, William G., M.D., Surgeon to the Presbyterian Hospital and to the Philadelphia Hospital.

1885. Potter, Thomas C., M.D.

1887. Price, Jacob, M.D.

1889. Price, Joseph, M.D., Physician-in-charge of the Preston Retreat and of the Female Department of the Philadelphia Dispensary.

ELECTED

1889. Randall, B. Alexander, M.D., Professor of Otology in the University of Pennsylvania and in the Philadelphia Polyclinic; Ophthalmic and Aural Surgeon to the Children's Hospital; Otologist to Rush Hospital.

1887. Reed, Charles H., M.D.

1885. Reichert, Edward T., M.D., Professor of Physiology in the University of Pennsylvania.

1888. Rex, George A., M.D.

1891. Rhoads, Edward G., M.D.

1891. Risley, S. D., M.D., Lecturer on Ophthalmology in the University of Pennsylvania; Attending Surgeon at the Wills Eye Hospital; Professor of Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine.

1882. Roberts, A. Sydney, M.D.

†1878. Roberts, John B., M.D., Professor of Anatomy and Surgery in the Philadelphia Polyclinic; Professor of Surgery in the Woman's Medical College of Pennsylvania; Surgeon to the Methodist Hospital.

1888. Robins, Robert P., M.D., Visiting Physician to the Dispensary of the House of Industry, to the Church Home for Children, and to the Board of Guardians of the Poor; Lecturer on Chemistry in the Episcopal Academy.

†1838. Ruschenberger, W. S. W., M.D., Medical Director, U. S. Navy.

*1864. Sargent, Winthrop, M.D., Roxbury, Mass.

†1866. Schaffer, Charles, M.D., Professor of Botany in the Pennsylvania Horticultural Society.

1887. De Schweinitz, George E., M.D., Clinical Professor of Ophthalmology in Jefferson Medical College; Professor of Ophthalmology in the Philadelphia Polyclinic; Ophthalmic and Aural Surgeon to the Children's Hospital; Ophthalmologist to the Orthopædic Hospital and to the Philadelphia Hospital; Consulting Ophthalmic Surgeon to the Methodist Episcopal Hospital.

ELECTED

1892. Seiss, Ralph W., M.D., Professor of Otology in the Philadelphia Polyclinic.

1888. Seltzer, Charles M., M.D.

1875. Seyfert, Theodore H., M.D.

1884. Shaffner, Charles, M.D., Ophthalmic Surgeon to the Presbyterian Hospital.

1887. Shakespeare, Edward O., M.D., Pathologist to the Philadelphia Hospital.

1876. Shippen, Edward, A.M., M.D., U. S. Navy (retired).

1891. Shober, John B., M.D., Surgeon to the University Hospital Dispensary and to the Gynecean Hospital Dispensary; Examining Surgeon for Pensions, Philadelphia.

1890. Shoemaker, George Erety, A.M., M.D., Visiting Surgeon to the Methodist Hospital; Surgeon to Out-patient Department of the Presbyterian Hospital.

†1893. Shoemaker, Harvey, M.D., Visiting Physician to The Sheltering Arms; Physician to Out-patient Department of the German, Episcopal, and Children's Hospitals.

1880. Simes, J. H. C., M.D., Professor of Genito-urinary and Venereal Diseases in the Philadelphia Polyclinic; Surgeon to St. Christopher's Hospital.

1873. Simpson, James, M.D., Physician to St. Mary's Hospital.

1872. Sinkler, Wharton, M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Neurologist to the Philadelphia Hospital; Physician to the Epileptic Hospital of Philadelphia.

*1863. Smith, A. K., M.D., U. S. A., New Hartford, Conn.

*1864. Smith, Edward A., M.D., New York.

1875. Starr, Louis, M.D., Physician to the Children's Hospital; Consulting Physician to the Maternity Hospital.

1892. Steinbach, Lewis W., M.D., Professor of Clinical and Operative Surgery in the Philadelphia Polyclinic; Visiting Surgeon to the Philadelphia Hospital and to the Jewish Hospital of Philadelphia.

ELECTED

1884. Stelwagon, Henry W., M.D., Ph.D., Clinical Professor of Dermatology in the Woman's Medical College; Clinical Professor of Dermatology in Jefferson Medical College; Dermatologist to the Philadelphia Hospital; Physician to the Department for Skin Diseases of the Howard Hospital and the Northern Dispensary.

1888. Stewart, David D., M.D., Clinical Lecturer on Medicine in Jefferson Medical College; Physician to St. Mary's Hospital and to St. Christopher's Hospital for Children.

†1842. Stillé, Alfred, M.D., LL.D., Professor (Emeritus) of the Theory and Practice of Medicine in the University of Pennsylvania; Consulting Physician to the Maternity Hospital and to the Woman's Hospital.

1846. Stocker, Anthony E., M.D.

1884. Stryker, Samuel S., M.D.

1886. Taylor, John Madison, M.D., Neurologist to Howard Hospital; Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Assistant Physician to the Children's Hospital; Professor of Children's Diseases in the Philadelphia Polyclinic; Clinical Professor of Diseases of Children in the Medico-Chirurgical College.

1867. Taylor, R. R., M.D.

1887. Taylor, William J., M.D., Surgeon to St. Agnes' Hospital, and Assistant Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases; Professor of Orthopædic Surgery in the Philadelphia Polyclinic.

1886. Taylor, William L., M.D., Instructor in Clinical Gynecology in the University of Pennsylvania, and Chief of the Clinic and Assistant Gynecologist to the Hospital of the same; Surgeon-in-chief to the Beacon Service for Women.

1867. Thomas, Charles H., M.D.

ELECTED

†1869. Thomson, William, M.D., Professor (Honorary) of Ophthalmology in the Jefferson Medical College, and Ophthalmic Surgeon to the Hospital of the same; Emeritus Surgeon to Wills Eye Hospital.

1866. Tyson, James, M.D., Professor of Clinical Medicine in the University of Pennsylvania; Physician to the University Hospital and to Rush Hospital for Consumption.

1864. Vandyke, E. B., M.D.

1873. Van Harlingen, Arthur, M.D., Professor of Diseases of the Skin in the Philadelphia Polyclinic; Dermatologist to the Howard Hospital.

1893. Vansant, Eugene Larue, M.D., Lecturer on Clinical Medicine in Jefferson Medical College; Visiting Physician to the Philadelphia Hospital.

1883. Vinton, Charles Harrod, M.D.

1885. Walker, James B., M.D., Attending Physician to the Philadelphia Hospital; Lecturer on Clinical Medicine and Consulting Physician to the Woman's Hospital.

1893. Warren, Joseph W., M.D., Associate Professor of Physiology in Bryn Mawr College.

1886. Watson, E. W., M.D., Physician to the Pennsylvania Institution for the Blind; Visiting Physician to the Home for Consumptives.

1875. Webb, William H., M.D.

1883. Welch, William M., M.D., Physician to the Municipal Hospital for Contagious Diseases; Lecturer on Exanthemata and Vaccinia in the Medico-Chirurgical College.

1893. Westcott, Thompson S., M.D., Physician to Dispensary for Diseases of Children and Hospital of the University of Pennsylvania.

1884. Wharton, Henry R., M.D., Demonstrator of Surgery in the University of Pennsylvania, and Assistant Surgeon to the Hospital of the University of Pennsylvania; Surgeon to the Children's, Presbyterian, and Methodist Hospitals.

ELECTED

1883. Whelen, Alfred, M.D.

1878. White, J. William, M.D., Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to the Maternity Hospital.

†1880. Willard, DeForest, M.D., Clinical Professor of Orthopædic Surgery in the University of Pennsylvania; Surgeon to the Presbyterian Hospital; Consulting Surgeon to the White and to the Colored Cripples' Homes and to the Home for Incurables.

*1878. Williamson, Jesse, M.D., Colorado Springs, Colorado.

1881. Wilson, H. Augustus, M.D., Professor of General and Orthopædic Surgery in the Philadelphia Polyclinic and College for Graduates in Medicine; Clinical Professor of Orthopædic Surgery in the Woman's Medical College of Pennsylvania: Clinical Professor of Orthopædic Surgery in Jefferson Medical College; Consulting Surgeon to the Kensington Hospital for Women.

1874. Wilson, James C., M.D., Professor of the Practice of Medicine and of Clinical Medicine in Jefferson Medical College, and Physician to the Hospital of the same (Faculty Staff); Physician to the German Hospital.

†1884. Wirgman, Charles, M.D., Physician to the Hospital of Jefferson Medical College and to the Howard Hospital; Physician to Out-patient Department of Children's Hospital.

1852. Wister, Owen Jones, M.D., Consulting Physician to the Jewish Hospital.

1893. Wolff, Lawrence, M.D., Demonstrator of Chemistry in the Jefferson Medical College; Visiting Physician to the German Hospital; Clinical Professor of Medicine in the Woman's Medical College.

1893. Wood, Alfred C., M.D., Assistant Surgeon to Gynecean Hospital; Instructor in Clinical Surgery in the University of Pennsylvania; Assistant Surgeon to the University Hospital; Surgeon to Out-patient Department of the University Hospital.

ELECTED

1865. Wood, Horatio C., M.D., Professor of Materia Medica, Pharmacy, and General Therapeutics in the University of Pennsylvania, and Clinical Professor of Diseases of the Nervous System in the Hospital of the same.

1880. Woodbury, Frank, M.D., Honorary Professor of Clinical Medicine in the Medico-Chirurgical College of Philadelphia, and Physician to the Hospital of the same.

1866. Woods, D. F., M.D., Physician to the Presbyterian Hospital.

1888. Woodward, Charles E., M.D., Physician to the Chester County Prison and West Chester Board of Health; U. S. Examining Surgeon.

1878. Wormley, Theodore G., M.D., LL.D., Professor of Chemistry in the University of Pennsylvania; U. S. Examining Surgeon.

1860. Wurts, Charles Stewart, M.D., Physician to Foster Home.

1861. Yarrow, Thomas J., M.D.

1889. Young, James K., M.D., Instructor in Orthopædic Surgery and Assistant Demonstrator of Surgery in the University of Pennsylvania; Orthopædic Surgeon in the Out-patient Department of the Hospital.

1887. Ziegler, Walter M. L., M.D., Assistant Aural Surgeon and Chief of the Dispensary for Diseases of the Ear in the Hospital of the University of Pennsylvania.

[It is particularly requested that any change of appointment, or any error in the titles of Fellows as published, may be communicated to the Committee of Publication before the first of November in each year, in order that the list may be made as nearly correct as possible.]

NECROLOGICAL LIST FOR 1893.

Fellows.

J. GIBBONS HUNT,	April 29, 1893.
JOHN C. HALL,	July, 4, 1893.
LEWIS K. BALDWIN,	August 6, 1893.
JOHN M. KEATING,	November 17, 189

Associate Fellow.

GEORGE C. SHATTUCK, March 22, 1893.

Resigned Fellowship.

OLIVER P. REX,	December 6, 1893.

ASSOCIATE FELLOWS.

[Limited to Fifty, of whom Twenty may be Foreigners.]

ELECTED

1873. Acland, Henry W., M.D., F.R.S., Oxford, England.
1890. Baccelli, Guido. Rome, Italy.
1877. Barnes, Robert, M.D., London, England.
1876. Billings, John S., M.D., U. S. A., Washington, D. C.
1886. Bowditch, Henry P., M.D., Boston, Massachusetts.
1877. Chaillé, Stanford E., M.D., New Orleans, Louisiana.
1886. Cheever, David W., M.D., Boston, Massachusetts.
1876. Comegys, C. G., M.D., Cincinnati, Ohio.
1876. Corson, Hiram, M.D., Norristown, Pennsylvania.
1893. Councilman, William T., M.D., Boston, Mass.
1876. Davis, N. S., M.D., Chicago, Illinois.
1886. Draper, William H., M.D., New York.
1892. Emmet, Thomas Addis, M.D., New York.
1883. Fayrer, Sir Joseph, M.D., LL.D., F.R.S., London, England.
1892. Fitz, Reginald H., M.D., Boston, Mass.
1876. Green, Traill, M.D., Easton, Pennsylvania.
1883. Heath, Christopher, F.R.C.S., London, England.
1892. Holmes, Oliver Wendell, M.D., Boston, Mass.
1874. Jackson, J. Hughlings, M.D., London, England.
1891. Jacobi, A., M.D., New York.
1893. v. Jaksch, Rudolf, M.D., Prague, Bohemia.
1876. Johnson, George, M.D., F.R.S., London, England.
1876. Jones, Joseph, M.D., New Orleans, Louisiana.
1893. Kerr, John G., M.D., Canton, China.
1877. Lister, Sir Joseph, Bart., M.D., LL.D., F.R.S., London, England.

ELECTED

1886. McGuire, Hunter, M.D., Richmond, Virginia.
1876. Moore, E. M., M.D., Rochester, New York.
1876. Mowry, R. B., M.D., Allegheny City, Pennsylvania.
1873. Ogle, John W., M.D., London, England.
1874. Paget, Sir James, Bart., M.D., LL.D., F.R.S., D.C.L., London, England.
1876. Pollock, A.M., M.D., Pittsburg, Pennsylvania.
1876. Porcher, F. Peyre, M.D., Charleston, South Carolina.
1886. Reeve, John C., M.D., Dayton, Ohio.
1886. Senn, Nicholas, M.D., Milwaukee, Wisconsin.
1886. Thomas, T. Gaillard, M.D., New York.
1869. Valcourt, Th. de, M.D., Cannes, France.
1892. Virchow, Rudolf, M.D., Berlin, Germany.
1892. Welch, William H., M.D., Baltimore, Maryland.
1886. Whittaker, James T., M.D., Cincinnati, Ohio.
1886. Yandell, David W., M.D., Louisville, Kentucky.

CORRESPONDING MEMBERS.

ELECTED

1880. Carrow, Flemming, M.D., United States.
1880. Chiara, Domenico, M.D., Florence, Italy.
1886. Dey, Kanny Lall, M.D., Calcutta, India.
1885. Rendu, Jean, M.D., Lyons, France.
1886. Richards, Vincent, Goalunda, India.
1889. Strahan, John, M.D., Belfast, Ireland.

CONTENTS.

PAGE

List of Officers and Standing Committees i
List of Presidents of the College ii
List of Fellows of the College iii
Necrological List for 1893 xxiv
List of Associate Fellows of the College xxv
List of Corresponding Members xxvi
Memoir of D. Hayes Agnew, M.D., LL.D. By J. William White, M.D. xxix
Notice of the Life and Character of George Cheyne Shattuck, M.D., an Associate Fellow of the College. By Alfred Stillé, M.D. lxvi
Annual Address of the President lxxvii
Additions to the Mütter Museum lxxxiii

Piperazin in the Treatment of Stone in the Kidney: Report of Cases. By David D. Stewart, M.D. 1
Neuritis and Myelitis, and the Forms of Paralysis and Pseudo-paralysis following Labor. By Charles K. Mills, M.D. 17
Symphysiotomy *versus* its Substitutes; with the Report of a Case of Symphysiotomy. By Charles P. Noble, M.D. 51
Report of a Case of Syringomyelia, with Exhibition of Sections of the Spinal Cord. By James Hendrie Lloyd, A.M., M.D. . . . 64
Exhibition of Specimens from Cases of Syringomyelia. By Wharton Sinkler, M.D. 80
A Second Case of Liver Abscess due to Disease of the Vermiform Appendix. By George Erety Shoemaker, M.D. 82
Treatment of Chronic Valvular Disease of the Heart. By James Tyson, M.D. 89
Peliosis Rheumatica in a Bleeder. By Thomas D. Dunn, M.D. . 100
A Case of Leukæmia with Rare Lymphoid Growths of Orbits and Parotid Glands. By Thomas D. Dunn, M.D. 103

PAGE

Some Results of a Bacteriological Examination of the Pipettes and Collyria taken from a Treatment Case used in Ophthalmic Practice, with the Effects of Inoculations. By G. E. de Schweinitz, M.D., and E. A. de Schweinitz, Ph.D. 110

The Etiology of Membranous Rhinitis (Rhinitis Fibrinosa). By A. C. Abbott, M.D. 122

Red Chromidrosis (?). By M. B. Hartzell, M.D. 133

A Study of the Influence of Chloroform on the Respiration and Circulation: A Contribution from the Laboratory of Experimental Therapeutics of the Jefferson Medical College of Philadelphia. Being a Report to the Government of His Highness the Nizam of Hyderabad. By H. A. Hare, M.D., and E. Q. Thornton, M.D. 137

Case of an Enormous Aneurism. By J. K. Mitchell, M.D. . . 178

Cleft Palate, both Hard and Soft, and Hare-lip. By W. S. Forbes, M.D. 181

Synchronous Amputation of Both Thighs for Railroad Injury. By Isaac Massey, M.D. 189

A Case of Well-modulated Voice Without Artificial Appliance, in a Patient from whom the Larynx and First Tracheal Ring have been Removed, and in whom Respiration is maintained Without a Canula. By J. Solis-Cohen, M.D. 191

Chloroform Anæsthesia. By H. C. Wood, M.D. 198

Exhibition of an Œsophageal Stethoscope, with Remarks on Intrathoracic Auscultation. By S. Solis-Cohen, M.D. . . . 218

Einhorn's Gastrodiaphane. Exhibited by S. Solis-Cohen, M.D. . 222

Operations for the Correction of Inveterate Pes Varus. By G. G. Davis, M.D., M.R.C.S. 225

Two Cæsarean Sections and Three Symphysiotomies: A Year's Work in the Surgical Treatment of Insuperably Obstructed Labor. By Barton Cooke Hirst, M.D. 235

Note on the Effect of Massage on the Blood-count. By John K. Mitchell, M.D. 240

Remarks upon the Comparative Difference between Certain Wounds Inflicted by the Projectiles of the Large and Small Calibre Hand Weapons. By Captain Louis A. La Garde, U. S. Army . 243

D. HAYES AGNEW, M.D., LL.D.

MEMOIR OF D. HAYES AGNEW, M.D., LL.D.

By J. WILLIAM WHITE, M.D.,
PROFESSOR OF CLINICAL SURGERY IN THE UNIVERSITY OF PENNSYLVANIA.

[Read January 4, 1893.]

In discharging the task which has been assigned to me by the President of the College, and which I regard as at once a duty and an honor, I do not propose to detain you with an account of the early life and work of Dr. Agnew, nor with many biographical dates or details. The excellent custom of embodying such information in the memoirs of distinguished Fellows may well be disregarded in this instance, as a biography of Dr. Agnew just published gives the main incidents of his long and useful career. I shall, accordingly, content myself with the briefest possible statement of the facts which seem essential in attempting to arrive at a just estimate of his position and rank in the profession of this country and among the surgeons of the world, and with a few reminiscences, in which I beg you to excuse occasional mention of my own views or experiences, for the reason that everything relating to him is of interest to hundreds, and is, therefore, worth recording.

He, himself, said not very long ago, in a sketch of his dear friend, Dr. Beadle, that "to preserve, in some tangible or permanent form, a record of the life-work of those who, after having achieved distinction in some one or more of the various spheres of human pursuits, have gone to swell the ranks of

the great silent majority is a custom no less commendable than beautiful."

Dr. Agnew was born in Lancaster County, November 24, 1818, of a family which, it is said, can be traced through many generations of North of Ireland and Scotch ancestry to Norman progenitors. In this country, for nearly two centuries, it has been prominent in the history of Pennsylvania, and especially in that of the county of his birth. He was educated at Moseow College, in Chester County, spent some time (1833-34), at Jefferson College, in Canonsburg, Pa., and a year (1834–35), at Delaware College, Newark, Del. He graduated in the medical department of the University of Pennsylvania in 1838, before he was twenty-one years of age. The first few years of his professional life were spent in practice in the vicinity of Nobleville and Pleasant Garden, Chester County.

During this time he married (1841), and, being invited to enter the business which had been left by his wife's father, he joined his brothers-in-law in the formation (1843) of the firm of Irwin & Agnew, iron founders. Fortunately this venture was unsuccessful. The firm failed in three years (1846), from causes associated with a general depression of the iron industry which occurred at that time, and from the absence in the locality of their works of sufficient facilities for the transportation of ore and fuel.

Dr. Agnew returned to the practice of medicine, and for two years (1846-48), practised in Chester and Lancaster counties. He was not content, however, with the future which opened up before him. His natural bent was toward the study and teaching of anatomy and surgery. In the country opportunities for dissection were obtained with the greatest difficulty, and there were not only no classes to whom to impart information, but no colleagues or co-workers with whom to discuss it. For these reasons, and probably because he had a consciousness, even though vague and unformulated, of the capacity for good work which lay within him, he came to Philadelphia, in 1848, with the purpose of making

it his permanent home and of prosecuting his studies in his favorite subjects.

I have always regarded this step of Dr. Agnew's with unmixed admiration. He was then thirty years of age. He had had seven years of country practice and three years of business experience. His life thus far had been a hard and laborious one; his disappointments must have been bitter, and, as regards his extra-professional essay, almost overwhelming. He had not only failed to accumulate any capital, but he had business debts which, to his sensitive and upright nature, seemed obligations which he was bound in honor to repay.

He left the region in which he had been born and reared, the friends of his family and of his early manhood, the patients who had learned to trust and depend upon him, and came to a city in which the profession was overcrowded, the competition for place and practice keen and unceasing, and the aspirants for both the honors and the rewards of medicine many and able. The move is, perhaps, the earliest traceable indication in his public life of the self-reliance and clearsightedness which came to be recognized as among his most marked characteristics.

After some deliberation he established himself in a large old-fashioned house at 16 N. Eleventh Street, where I first saw him when, as a boy of ten, I took a note to him from my father, asking him to see a surgical case in a member of our family. This was in 1860. For twelve years he had been laying the foundations of his future success. In 1852 he had begun his teaching of practical anatomy and operative surgery at the Philadelphia School of Anatomy, and his name is still the most distinguished in the long list of able men who have been connected with that institution. In 1854 he had been elected a surgeon to the Philadelphia Hospital, and had thus been given not only his first hospital wards, but also his first opportunity to perfect himself in the teaching of clinical surgery. He always regarded this step as one of the most important of his professional life, and once in speaking of it to me, said he had already, at that time, decided that he

"might as well attempt to be a gardener without a garden as surgeon without a hospital." He had also, for two years, been occasionally substituting for Dr. Henry H. Smith, professor of surgery at the University, in giving clinical instructions to the students. At the time of which I speak (1860) his reputation as an able operator and a sound consultant was firmly established and rapidly spreading, but his practice was not yet, as he told me, what would now be called a lucrative one. He was, however, only forty-two years of age, in the enjoyment of the fullest mental and physical vigor, and undoubtedly saw success within his grasp. I remember well the kindly manner in which he received me, and the promptness with which he left the fire, before which he had been reading, changed his slippers for his shoes, and prepared to answer the call which I had brought, although it was late on a disagreeable evening He had then, as always, an impressive personality and a magnetism felt by most persons who came within the sphere of his influence. It was not of the sort that is exploited in the journals as characterizing brilliant politicians and successful criminal lawyers, nor yet of the variety which history teaches us has been possessed by many great statesmen and victorious generals. If I attempted to epitomize or explain the secret of Dr. Agnew's attractiveness to so many and such different classes of people, I would say that "kindliness" expressed it perhaps better than any other word. There were strength and energy and determination back of it, with a basis of broad knowledge and justifiable self-confidence; but, all the same, the chief impression he made was of a tender benevolence which always regarded the feelings and interests of others, and which pervaded his whole atmosphere. It was noticeable then even to a child, and it grew as he advanced in years, until the title of the "Dear Old Man," which was given him by the younger Gross, was universally accepted as in the highest degree applicable.

During the civil war Dr. Agnew had large opportunities—chiefly at the Hestonville General Hospital—for operative work, and it is needless to say profited by them to the utmost.

In 1863 he was elected Surgeon to Wills Eye Hospital; in 1865, Surgeon to the Pennsylvania Hospital; in 1867, Surgeon to the Orthopædie Hospital; in 1870, Professor of Clinieal Surgery, and in 1871 Professor of Surgery in the University of Pennsylvania, retaining the latter positions until 1889, when he resigned them to be created Emeritus Professor of Surgery, and Honorary Professor of Clinical Surgery.

During all these years he grew steadily in professional strength: his clinics were crowded, his office was filled, his services were in daily demand in all parts of the Middle States, and the University of Pennsylvania was sending out, year after year, hundreds of young men who regarded him, and justly, as the best possible adviser in all eases of surgieal disease or injury.

His election in 1889 to the Presideney of the College of Physieians was a distinetion which, like all the others, came to him unsought and unsolieited, but which he nevertheless highly appreeiated. It resulted from the very widespread feeling in the College that he should not be permitted to end his days without adding his name to the list of those gentlemen who, without exeeption, have for years reciproeally honored and been honored by this organization, perhaps the most conservative in America.

My personal remembranees of him begin again in the winter of 1868, when, twice a week, after supper I used to hurry back to the University to get a front seat at his half-past seven o'cloek leetures on surgieal anatomy. He was then probably the most popular teacher at the University, and deservedly so. I certainly have never heard lectures on anatomy which, for clearness of description, actual teaehing foree and living interest, could compare with those whieh he then gave. On those two nights the room was crowded with the students of both elasses, and, as he always spent the latter part of the evening in the dissecting-room, every subjeet was surrounded by its full quota of diligent workers, who, if I may judge others by myself, found the chief attraction in the chance of getting a pleasant word of advice or instruction from him.

He looked then very much as he did for the next twenty years of his life. His hair was already thinning and beginning to turn white, as was the moustache, which was prolonged on the cheeks in a military fashion. His height, of more than six feet, and his fine muscular development, made his figure commanding in spite of the slight professional stoop which he always had, the result of hours spent over sick-beds and operating-tables. His blue eyes were keen but kind in their expression.

An old blue dress-coat with brass buttons, which he wore to these lectures, gave him, I remember, to my imagination, a military air, and this fancy came back to me in one of his last attacks of illness, when I found him sitting up in bed with a handkerchief tied around his forehead on account of a severe supra-orbital neuralgia. He looked like a wounded grenadier. I told him of my boyish idea about the blue coat, which he recalled, and said smilingly he would hardly venture to wear it before a medical class of the present day, in spite of their alleged improvement in manners. He has twice in my presence alluded to himself, half laughingly, half seriously, as "homely." Homely he was in the sense in which Chaucer and most English people at the present day employ the word, that is, he was domestic in habits and simple and affable in manners; but homely in our perverted American signification, which usually implies not only plainness of feature but positive ugliness, he never was. A cold-blooded critical analysis of his features might disclose some reason for such an opinion, but no one who knew him looked at Dr. Agnew in that spirit. Goodness and kindness of character shine so clearly through some faces that defects or irregularities are forgotten, and his was one of them.

The College of Physicians and the University are fortunate in possessing masterly portraits of him, which will help to bring before future generations of Fellows and of students his striking personality. We of the present day need no such reminders. We carry his likeness in our hearts.

In reviewing his life one is struck with the fact that both

hereditary and personal influences were such as favored the growth and development of that side of his character upon which, as fate willed it, the greatest demand was to be made in years to come. The strain of Scotch blood brought with it dogged perseverance, enduring patience, disregard of luxury, even of personal comfort, ability to sustain uncomplainingly the reverses of fortune, and to submit contentedly to the long-continued economies which they necessitated. But it must be added that these virtues, so often associated with a sombre or stern disposition, were in his case tempered by geniality, cheerfulness and an unvarying and all-embracing tolerance that was one of his chief characteristics.

The wholesome out-door life which he led in the country for so long a time was the best sort of "physical culture," and doubtless those years, which he sometimes thought had been in a sense wasted, came back to him in the form of increased energy, endurance, and good health later in life. His very misfortunes supplied an additional stimulus to a character which naturally was so domestic and affectionate that ambition alone might not have brought about its transplantation from the familiar soil in which it had been reared and nourished.

It is a trite observation that our apparent trials often prove to be our greatest blessings. There can be little doubt that Dr. Agnew contained within him the qualities which would have commanded success, even under the unfavorable conditions associated with material prosperity, but neither is it doubtful that the years spent while waiting for practice, when he went to the dissecting-room after his morning office hours, went back after his mid-day dinner, spent most of the afternoon there and returned after an early supper, to leave only when the last student had gone and the lights were extinguished, were the years during which he laid broad and deep the foundations of his success. They were years, also, the reward of which was not postponed to a future period. Dr. Agnew has often told me that on the whole he regarded the days spent in the rooms on Chant Street, the "Windmill Street" of

Philadelphia, as the happiest days of his life, and he once at least made the same statement in public.

When we come to estimate the true value, and the real and enduring position of one of our profession who has left us, we can, perhaps, adopt no better gauge than the opinion of his pupils. History shows us that almost invariably the surgical contemporaries of a great surgeon, with whatever affection they may have cherished him, were still, in a sense, his rivals; they learned, or failed to learn, at the same time, or by the same stern experiences; they often differed, and, being human, when they chanced to be right derived an increased pleasure from the fact that he was wrong; their estimate, in other words, was often unconsciously tinctured by personal prejudice, or influenced by motives not apparent to themselves. Who would to-day be content to accept Cline's opinion of Sir Astley Cooper, or Fergusson's of Syme, or Syme's of Liston, or Pott's of John Hunter?

Dr. Agnew, more than any man of equal eminence in our profession with whose history I am familiar, was free from this sort of belittling animosity toward others, and, perhaps as a consequence, rarely if ever excited it. But when one dies at seventy-four, after more than a half-century of professional work, few of his own years are left to testify, and still fewer are apt to place on record their final and deliberate judgment.

The laity, however sincere their admiration and however well founded it may sometimes prove, are essentially unfit to pronounce upon the claims of medical men to either present or posthumous honors. They are too apt to mistake show for brilliancy, self-assertion for force of character, and notoriety for fame. In Dr. Agnew's case they held him at his just value but their verdict can never be regarded as final or conclusive.

It is, after all, in the case of great surgeons at any rate, upon the testimony of their pupils that their ultimate place in the history of our profession depends. Nearly every surgeon who has made his name immortal has been a teacher, and usually as great a teacher as he was a surgeon. For one exception

like John Hunter, a hundred illustrious names could be adduced to emphasize the truth of this statement. Their lives have been written, their merits described, their achievements recorded, their virtues extolled by those whom they have taught and guided, and Dr. Agnew constitutes no exception.

From the length and breadth of this land the pupils whom for ten years (1852-62) he instructed in the Philadelphia School of Anatomy, and those who during the next thirty years had the paths of surgical science made easy for them in the University of Pennsylvania by his wise, clear, practical teachings, have over and over again testified to their respect for him living and their reverence for him dead. Their estimate, their decision, will be those accepted by future generations, and upon them Dr. Agnew's fame will rest justly and securely.

This professional reputation, thus testified to, will be found to depend chiefly upon the following factors: (1) The clearness of his teachings; (2) the soundness of his judgment; (3) the precision of his operations; (4) the character of his writings.

Of course, some of his success was due to the personal qualities which have already been mentioned. A man who cannot enter a household or a sick-room without diffusing about him the almost indescribable sense of repose and confidence that he inspired, the feeling on the part of the anxious or grief-stricken relatives that however the case is going everything humanly possible is being done for the patient, and on that of the medical attendant that exhaustive consideration has been given to every detail of the treatment: such a man, I say, is certain of increasing practice as soon as he has made a beginning and has become known to the public and the profession. Something of this quality is occasionally inherent in men whose knowledge and natural capacity do not justify it. The public is then deceived for a time, but the profession rarely or never. To the degree in which it was possessed by Dr. Agnew it may safely be said it is never found except in

association with wide experience, broad professional culture in the best sense of the word, and pre-eminent ability. In its effect upon the acquiring of practice it is perhaps more important than any of the factors above mentioned, but the reputation which it brings is evanescent, and when years have elapsed it will be found, as I have said, that the surgeon will be known to posterity, if known at all, as a teacher or writer, a consultant or operator, not simply as a successful practitioner.

(1) As a teacher he was remarkable for his simple, plain, straightforward methods, his entire disregard of oratorical effort, his faculty of making clear and easily comprehensible even the abstruse and complicated portions of his subject. He never spent time or labor upon mere eloquence, but was always so earnest and so obviously desirous of conveying information as to hold the attention of his class, and so concise and practical as to leave his teachings indelibly impressed upon their memories. His views were so evidently the outcome of mature experience, and so stamped with the seal of honest conviction, that his enunciation of them gained added impressiveness and made his course upon surgery not only one of the most useful, but also one of the most pleasant, of their University experiences, to thousands of undergraduates. Without apparent effort, and with a skill born of thorough knowledge and perfect mastery of his theme, each subject was presented to the student so clearly, simply, and directly, that it remained a part of his medical knowledge. His lectures were very uniform in quality, but I recall with especial admiration those upon hernia, upon fractures, and upon the ligation of arteries. To my mind they were models of what surgical teaching should be. I have heard the same opinion expressed many times by alumni who preceded and by those who followed me at the University, and neither my reading nor my experience leads me to believe that those lectures would suffer by comparison with the teachings of the greatest masters of our art in this or any previous epoch.

His habit at his clinic was to precede his operation by a

brief statement of the history of the case, and by some remarks upon diagnosis and prognosis, and upon his reasons for choosing the particular operative method he was about to employ. During the operation itself he said but little. Afterward he would usually leave some of the final details, the arrest of hemorrhage, insertion of sutures, application of dressings, etc., to his assistant, while he explained more fully what he had just done, and described what he had observed.

He possessed to a remarkable degree the power of interweaving with his admirable descriptions of surgical diseases and injuries the underlying anatomical facts upon which accurate diagnosis and successful treatment so commonly depend. Indeed, as a practical surgical anatomist he was in this country without a peer, and much of his success arose from his well-earned reputation in this department.

He was a natural teacher, always ready and pleased to impart information even outside of his lectures to anyone seeking it in the proper spirit, and did so in the clearest and most agreeable manner possible, making the dullest subject seem interesting, and investing it with a more extended significance by some illustrative or cognate fact.

He never, however, forgot the interests of his patients in his desire to convey instruction. I held the staff for him for some time and in a great many lithotomies before I had much experience in cutting for stone myself. He knew this, but as he did not believe in a multiplicity of fingers in wounds, or in removing the finger in these cases until the forceps were in the bladder, he never but once asked me to explore that organ prior to the removal of the calculus. On that occasion we were operating in a little town in the southern part of Delaware. After making the usual incision and putting his finger in the bladder, he spent a long time in exploring the *bas-fond* and the post-prostatic pouch in search of the stone which we had both heard and touched with the staff. Presently he asked me to slip my finger in as he withdrew his, as the condition was so extraordinary that he would like me to feel it.

The stone was swinging like a pendulum from the summit of the bladder, held there by a band of organized lymph quite firm and resistant.

The case was unique in his experience.

(2) As a consultant and as a practitioner perhaps his most noteworthy quality was the soundness of his judgment. Partly from his natural clearness of mental vision and good common sense, partly from his thorough preparatory training and his diligent and incessant study of everything that bore upon the teaching and practice of all departments of surgery, and partly, no doubt, from the enormous experience which in later years he could utilize for his guidance, he made singularly few mistakes in either the diagnosis or the treatment of surgical conditions.

It is given to none of us to be infallible; error and humanity are now, as always, inseparable; perfect wisdom and complete knowledge in even the narrowest of human occupations are unattainable. I desire to make no exaggerated claim for the subject of this memoir, but I feel sure that at least to this audience, which numbers so many who looked upon him as the court of last appeal in all surgical matters, I may venture to state my belief that few surgeons have ever lived who, in the presence of an obscure case, were so uniformly correct in their estimate of the precise diagnostic value of a surgical symptom, so accurate in their application of the general principles bearing upon the condition in question, and so conversant with all the varied possibilities which such cases present. His ability to unravel the tangled web of disease, to solve its intricate problems, seemed to me in the earlier days of my association with him to be little short of inspiration; later I came to recognize it as the legitimate outcome of keen observation, wide experience, and logical reasoning.

I have often thought that of all the great surgeons with whose lives I was familiar I could trace the closest resemblance between both the personal and the professional characteristics of Sir Astley Cooper and those of Dr. Agnew. I once said this to him, and he replied that while there was no surgeon whom

he would rather be thought to resemble, he feared that, like many other attempted parallels from the days of Plutarch until now, the facts would have to be made to conform to the theory.

And yet I may ask those of you who knew him best if the following description of the great English surgeon by his nephew might not have been written *verbatim et literatim* of Dr. Agnew:

"His influence did not arise from his published works, nor from his being a lecturer, nor, indeed, from any public situation which he held, although each of these circumstances had its share in producing the result; but it seemed to originate more from his innate love of his profession, his extreme zeal in all that concerned it, and his honest desire, as well as great power, to communicate his knowledge to another, without at the same time exposing the ignorance of his listener on the subject even to himself. This must be looked upon as one great cause why his public character became so much diffused by his professional brethren, for he owed little of his advancement in life to patronage. Another peculiar quality which proved always a great source of advantage to him was his thorough confidence in respect to his professional knowledge, so that after he had once examined a case he cared but little who was to give a further surgical opinion upon it. This must inevitably have instilled an equal degree of confidence in those consulting him."

To the younger men, and especially to the younger operators in the profession, he was more than a consultant. How many reputations he has helped to make by the encouragement and guidance which he never refused; how many more he has saved from wreck by the protection and support which his unassailable position permitted him to give to those whose ignorance, rashness, or ill luck got them into surgical difficulties, no one but himself ever knew. In suits for malpractice he was the bulwark and safeguard of dozens of unfortunate medical defendants. He always saw clearly just what could be truthfully said in explanation or extenuation of a bad result; and although in his evidence in such cases he was never

known to depart from the strictest veracity, he would probably have himself pleaded guilty to *suppressio veri* in more than one instance. What was said of Sir William Fergusson was true of him:

"Full of experience himself, and able to wield his powers so brilliantly, he was tender as to the failure of others, and nothing gave him more pleasure than to have an opportunity of helping someone out of difficulty, while the manner in which he gave this help was as graceful as the assistance itself was valuable."

One source of Dr. Agnew's power as a diagnostician was his very retentive memory for even the details of cases which he had once seen. No clinical observation, even of times long past, escaped him, and as for many years he was the chief consultant of three States, with a population of millions, his experience, almost without exception, furnished a parallel to the rarest and most obscure conditions. I have more than once begun to describe to him some recent experience or observation of my own, which seemed to me unique, only to have him interrupt me kindly and smilingly, and finish the description as accurately as if he had been present and had seen the case himself. Often, too, to my great profit, he would volunteer some words of explanation that instantly made clear all that was previously doubtful.

His own powers of observation were so well trained that in many surgical conditions a glance sufficed for a diagnosis, and although usually cautious about expressing an opinion, he would sometimes venture upon a positive one the moment he saw the case. This was notably so in patients with complete scrotal hernias or with large hydroceles, the distinction between which he made by the relation of the tumor to the anterior plane of the body; in cases of shoulder injury, where the position of the patient revealed the presence or absence of luxation; in deep abscesses, where a glance at the overlying skin and a touch with his opposing fingers completed his examination; in fracture near joints and in ununited fracture, where he almost seemed to have a special sense for deter-

mining preternatural mobility, and in many other classes of cases.

The story which Vidal de Cassis tells of Dupuytren, that within a few days of his death he was asked his opinion of a lesion of the elbow in a patient who was brought to his bedside, and without touching him diagnosed a luxation, which had been denied by an eminent colleague, but was subsequently demonstrated and reduced, might well have been told of Agnew.

(3) In speaking of his operative work I have purposely used the word precision to characterize it, instead of the usual eulogistic term—brilliancy. Dr. Agnew himself disliked the latter word, and with it the kind of operating which it often described, in which everything is sacrificed for the sake of what in athletics would be called a "record." He has more than once told me interesting anecdotes of the difficulties into which this habit of what he called "slap-dash" work had led some of his contemporaries. He agreed with my friend Mr. Treves, himself an exceptionally able operator, who says that the days of the so-called "brilliant" surgeon are over, and that it is no longer a matter of primary importance that a stone should be extracted, or a vessel tied, or a limb removed, in a limited number of seconds.

Dr. Agnew was naturally a quick operator, and his training dated back to the days when pre-anæsthetic traditions kept alive the theory that rapid surgery was good surgery, but he was, above everything else, a safe operator, a precise operator. His knowledge of anatomy included not only that of the dissecting-room, which, important as it is, is but the beginning for the surgeon, but that of the operating-table, which is often very different. He knew not only what the relations of parts and structures ought to be, but what they were in any particular case. He knew, in other words, not only the ordinary topographical anatomy of books, of the cadaver, and of the lecture-room, which is knowledge common to many, but also the anatomy of the individual upon whom he was operating, and of the disease which necessitated his interference. He

has often said to me that no two cases of hernia were precisely alike in their anatomical appearances; that each dissection of the neck for the removal of a new growth had a certain individuality which enabled him to remember it for years; and that one might cut through the loin twenty times to approach the colon or kidney, and find no two of the cases precisely identical in thickness of muscular layers, amount of peri-renal fat, etc.

It was in this knowledge of the conditions and appearances to be expected in a given case, the knowledge of these infinite variations, that he was far superior to any operator with whom I have had a chance to compare him. With the splendid foundation of practical anatomy which his years of teaching and demonstration had given him, his constant operating had developed in him this faculty of quick and certain recognition of the structures exposed to view or to touch, which gave his work the character of accuracy and precision that impressed everyone who saw it.

His use of all instruments was light but firm, devoid of flourishes or attempts at show, but strikingly graceful. His hand was very steady up to the last month of his life. His ability to work with either hand, although he did not himself consider it of much practical value, was certainly a great convenience, and was a peculiar and striking feature in his operating. He acquired it as a consequence of a severe onychia of the right index finger, which destroyed the matrix and which deprived him for a long time of the use of that hand, during which period he taught himself the use of the other.

The general level of his operative work was so high that it is difficult to select any particular cases or groups of cases for special mention, but possibly his peculiar abilities were made most manifest during the removal of a deep-seated growth of the neck or of a mammary tumor, the ligation of a large bloodvessel, the extraction of a vesical calculus, or the excision of a bone like the superior maxilla, or a joint like the knee or elbow.

One of his most marked characteristics was his imperturbability, described by his former colleague, Dr. Osler, with eloquent reference to its applicability to Agnew as "coolness and presence of mind under all circumstances, calmness amid the storm, clearness of judgment in moments of grave peril; a quality which in its full development has the nature of a divine gift, a blessing to the possessor, a comfort to all who come in contact with him." This quality Dr. Agnew had to such a degree that nothing that could happen during an operation, however unexpected or undesirable, seemed greatly to disturb him. Terrific and unlooked-for hemorrhage, profound shock, alarming collapse, threatened asphyxia, all possible surgical accidents and emergencies, were embraced within his experience, and I have seen him meet each of them in the same prompt, quiet, masterful manner which assured and gave confidence to everyone who was working with him. Even death on the operating-table has come to him without disturbing his equanimity. He had it occur once during tracheotomy in a desperate case, but it was many years ago.

The only other time I know of, and the only time I ever saw Dr. Agnew lose a patient on the table, was during an operation for hemorrhoids in 1887 at the Bingham House in this city. The patient, who was apparently well and strong, had taken less than four ounces of ether, and was breathing well when the first ligature was tightened. His respiration immediately stopped, and although his heart continued to beat for nearly half an hour he died without making even an attempt at respiration. The autopsy showed an apoplexy into the floor of the fourth ventricle. That half-hour was one of the longest in my life. The physical exertions employed in the efforts at resuscitation were in themselves exhausting, and in addition there was the mental distress incident to such a sudden and unexpected termination of a minor case. At the end I was drenched with perspiration, unnerved from excitement and disappointment, and felt ten years older. Dr. Agnew was as cool and placid as if the occurrence was a common one in his experience. As we walked down to our carriages on

Eleventh Street he made a remark which I have never forgotten. He said: "I had hoped to escape the accident of an ether death, but it occurs once in a certain number of thousands of cases in the best hands, and I've no doubt I passed my limit long ago. We were not to blame." Then after he got in his carriage he leaned out to say: "If you are asked about this, just say it was my case. I'll accept all the responsibility."

Philosophical and unchanged in the presence of calamity, tender and thoughtful of the interests of others, he was then, as always, one man in ten thousand.

Dr. Agnew was imperturbable not merely as an operator, but in all the ordinary relations of life. No surgeon ever lived and attained eminence who was more careful to throw around his patients every safeguard, or who strove more earnestly after successful results; and yet no surgeon accepted untoward results with greater equanimity when it became apparent that they were inevitable, or when the case had ended. His mental attitude under these circumstances seemed to be that of a consistent fatalist, and I have many times envied him the unruffled placidity with which he finally learned of the unfavorable termination which he had, perhaps, contested hour by hour for long and anxious days. He used to explain it by saying that he felt that he had done his best and could do no more; but he probably had the consolation denied to most of us of knowing that *his* best was *the* best, although he was too modest so to express it.

His patience as an operator was equally remarkable. I do not remember, as his assistant, or indeed in any other capacity, ever to have received a harsh or unkind word from him for faults of either omission or commission, although I must often have deserved it. The one thing that used to elicit from him an expression of impatience was the breaking of an important stitch during a staphylorrhaphy or an operation for vesico-vaginal fistula. He would then come as near to swearing as his principles permitted. One of the first private operations I ever saw him perform was for the relief of such a fistula in

the wife of a clergyman. The most troublesome stitch broke while he was tightening it, eliciting an ejaculation which led Dr. Elwood Wilson, who was present, to say, "Agnew, I'll tell Beadle on you!" referring to the Rev. Dr. Beadle, one of the most admirable and lovable of characters, and then, probably, Dr. Agnew's dearest friend. This raised a laugh, in which Dr. Agnew joined, and the stitch was reinserted. The incident represents the full extent to which he ever permitted himself to show temper, and I must confess that it was a source of gratification to me whenever a similar occurrence took place, as it momentarily diminished my sense of inferiority.

(4) His collected writings embrace a number of miscellaneous articles—important contributions to the surgery of lacerated perineum and vesico-vaginal fistula; papers on general surgical diagnosis, etc.—but the work of his life as a writer is his great *Treatise on the Principles and Practice of Surgery*, the three successive volumes of which bear the dates 1878, 1881, 1883.

It is safe to say that this magnificent monument to the learning, skill, and industry of one man will remain unrivalled in surgical literature. It is not likely that there will ever again be anyone who will combine the enormous experience, embracing every department of surgery, the clear judicial intellect, and the patient, untiring energy which enabled him in hours stolen from his family, from social pleasures, and from much-needed rest, to produce this remarkable exposition of his work and views.

It was, as has been said, his child, the dearly-beloved offspring of his brain, and while I regret that in the swift march of surgical science it must, if left without revision, inevitably fall into disuse, I can understand the sentiment which desires to keep it unaltered and unchanged as the most eloquent of all records of his splendid achievements.

And yet there are portions, and large portions, of the book which it seems to me can never grow surgically old or useless. Our successors may be too hurried to read the abstracts of the history of important surgical procedures, which, with infinite

labor and painstaking, he had conscientiously compiled; his pathology may become antiquated, and his therapeutic measures come to be looked upon as are those of Paré or Wiseman, but his admirable clinical descriptions, his comprehensive and well-balanced consideration of diagnostic points, his clear explanation of the surgical anatomy of disease, injury, and operation, must always remain as at present, a mine of information for the busy practitioner.

After many years of what may be called intimate acquaintance with the book I venture to assert, and I doubt not many of you will corroborate me, that in the directions I have indicated one may turn to it for help and guidance with more certainty of finding what he seeks than to any other single treatise on surgery in his library. This statement was made in some of the early reviews of the work, and I was once present when a surgeon of distinction from a neighboring city expressed the same opinion to Dr. Agnew. I remember well the simple, unaffected pleasure which it evidently gave him, and he afterward told me that he hoped it was true, as he regarded it as a higher compliment than any other that could be paid to a medical work.

The book went to a second edition in 1889, and is well deserving of many future editions. It is the property of the Trustees of the University of Pennsylvania.

He was not a conspicuously original surgeon. He invented a number of instruments and splints, notably that for use in transverse fracture of the patella; introduced some new operations, such as that for webbed fingers; modified others, as the musculo-cutaneous flap method in amputation and lateral lithotomy in children, and made many minor improvements in both surgical apparatus and operative technique. His claim to imperishable fame does not, however, rest upon any of his inventions, nor upon any one great addition to surgical science. It is not given to everyone to be a discoverer. Dr. Agnew had not a mind of the inventive, speculative, restless type which contributes the newest, but not always the most useful, ideas to our profession. Neither did his training nor his cir-

cumstances fit him for the plodding laboratory work, experimental and microscopic, as a result of which a Pasteur or a Lister or a Koch electrifies the world with some grand contribution to its knowledge. Dr. Agnew had a judicial mind, wonderfully fitted to weigh and decide upon the suggestions that came to it, and he had extraordinary mechanical and physical ability to carry into effect in an operative way whatever appealed to his deliberate judgment. As was said of Abraham Colles: "He possessed the art of touching briefly on the salient point of his cases, and was gifted with what Dove called 'the incomprehensible talent' of separating the essential from the immaterial in complicated phenomena."

He was eminently and above all others of his time—I am almost tempted to say of *all* times—a sound surgeon, a safe surgeon. One might be sure invariably that his opinion in a given case, even if it were not finally proven to be the right one, was well founded, and would stand creditably the test of adverse criticism; and in every case and under all circumstances it was equally certain that the opinion was given with the single idea of being of use to the physician or patient who had consulted him.

In special departments of surgery he has probably been excelled by not a few; but taking the whole round, including not only general and operative surgery, but gynecology, ophthalmology, genito-urinary surgery, syphilography, orthopædies, etc., he attained a degree of eminence which has rarely, if ever, been equalled, and to which our own times and generation furnish no parallel.

I have never seen or known of any instance in which he seemed in the least degree influenced by the temptations which constantly beset those of us who as teachers need clinical material, as operators desire to attempt new or hitherto untried procedures, as practitioners feel the universal need of money and of prestige. Since the death of Dr. Charles Hunter, his previous assistant, which was a great loss to the profession, to the University, and to Dr. Agnew himself, I was intimately associated with him in both his public and private

work, and for the last few years he did me the honor of referring to me those patients who required operation. I may, therefore, claim to have had every possible opportunity of observing and criticising the standard of his conduct in this respect, and I cannot conceive of a loftier or more unswerving devotion to the highest and best principles than that which he always displayed. Certainly no one ever brought to any occupation a more profound sense of its grandeur and nobility, a higher estimate of its relations to mankind, or a more steadfast purpose to consecrate his whole life to it than did Dr. Agnew to the study and practice of surgery.

For a long time he had a medical practice which was among the largest in Philadelphia. He retained it at first because he believed that while it was possible to be a good physician with no surgical experience whatever, he did not think that the converse was true, but always held that a surgeon was better and stronger in his own department if he had to watch and study and care for the medical conditions which are liable at any time before or after operation to complicate his purely surgical cases. Later, he had the not unusual experience of finding it next to impossible to refuse to see such cases in families who had for years been accustomed to depend upon him.

His therapeutics, both in medicine and surgery, were very simple, and I have found, and still find, among the prescriptions brought to me by his patients, astonishingly little variety. Bransby Cooper remarks of his distinguished uncle: "So simple were Mr. Cooper's prescriptions that he had five or six formulæ, which, under ordinary circumstances, constituted his complete pharmacopœia, and such medicines he kept constantly made up for the benefit of the poor."

Sir Joseph Lister once told me that during his very earliest days in Edinburgh, when he was still uncertain whether to remain there or to begin his work elsewhere, he consulted Mr. Syme. The latter told him that he would probably do well to stay there, but remarked that it really seemed as though there were not much left to do in the way of advancing

surgical science: little thinking at the time that the young man he was talking to, his future son-in-law, would almost alone and unaided effect the greatest revolution in surgery, and bring about the greatest step in advance which has been made since Harvey discovered the circulation of the blood.

Dr. Agnew never, to my knowledge, wavered in his profound and unvarying faith in the future of surgery. He would have had far more reason than Mr. Syme for believing that the limit of possible advance had been reached, as he had seen the introduction not only of anæsthesia, but of antisepsis. It was contrary, however, to his buoyant and hopeful disposition, and inconsistent with his own progressive spirit, to take this view of the science which had shown such infinite capacity for development. He believed, for example, that just as surgical tuberculosis is slowly but surely coming under the control of the surgeon, so carcinoma, the present opprobrium of surgery, would in time yield to methods of treatment yet to be discovered. He was always among the first to test new surgical procedures and to adopt them if they seemed to him consistent with fundamental principles and common sense. At a time when many of his juniors were skeptical as to the merits of antisepsis, or were even openly antagonistic, he gave it a thorough trial, and at once discarded in its favor the methods which he had employed for forty years. He was among the first to advocate the extension of the field of operative interference in fractures of the skull, one of the innumerable advances which antisepsis made possible. He performed nephrectomy and laryngectomy, supra-pubic lithotomy and prostatectomy, nerve-grafting, and other operations which were popularized only after he had reached an age when, upon many minds, a new idea has the effect of a foreign body upon the grosser tissues, and is either encapsulated and disappears or sets up irritation and is extruded. While Sir Henry Thompson was impeding the acceptance of litholapaxy by the profession in Great Britain, Dr. Agnew, although one of the most accomplished and successful of lithotomists, was employing it in the majority of his cases of calculus. He attacked brain tumors,

tried the modern methods for the radical cure of hernia (though he had previously devised, employed, and cast aside as useless a method of his own), removed the appendix both during attacks of inflammation and in the interval, employed the most recent technique in the treatment of ununited fracture, and in every direction kept fully abreast of the times up to the very week of his death.

On the other hand, he could not be induced to attempt operations which his judgment did not approve, and his last important paper, a judicial review of the results obtained by local surgeons in various forms of cerebral disease and injury, was written partly with a view of establishing the uselessness of certain procedures which he believed to be unphilosophical and unjustifiable. He thought the evidence showed that he was correct; but, if future experience had demonstrated the contrary to be true, it is safe to say that he would have been one of the first to admit it. I have already alluded to his behavior in reference to his own operation for the cure of hernia. A still more striking illustration, however, of his fair-mindedness is to be found in his attitude toward a question of public policy which, on account of the apparent conflict between hygiene and morals involved, has always excited the bitterest contention. I allude to the so-called "Contagious Diseases Acts," the recognition and regulation of prostitution by the State. Everything in Dr. Agnew's religious training and association, and many elements of his personal character, tended to lead him into the ranks of the opposition, where are to be found in every country where the subject has been discussed the large majority of the clergy and a small minority of our profession. In 1882, however, when he was sixty-four years of age, he wrote: "At one time I was disposed to take sides with the opponents of prostitution laws; but on a more comprehensive and careful study of the subject, and especially of the results which have been reached in those countries where such legal regulations are in operation, I am forced to believe that the evil is one which comes legitimately within the province of civil law."

To my mind this statement is one of the most interesting examples of the many which might be adduced in evidence of Dr. Agnew's broad-mindedness and absolute independence of thought and judgment.

It is obvious that Dr. Agnew's physical endurance must have been extraordinary to enable him to carry on at one time the teaching, writing, operating, and consulting, any one of which would have been a heavy burden for an average man. He told me not more than five years ago that he did not know what it was to be tired. One of the best illustrations of both his strength and his devotion to duty is afforded by an experience which he has more than once mentioned to me. He was called by telegram early one morning to an out-of-the-way place in the northern part of the State. He ate a hurried breakfast, caught his train, and by dint of close railway connections and a final drive of eight miles over a rough road reached the house of his patient late in the afternoon. An operation was required, and he performed it at once, a meal being prepared for him in the meanwhile. On looking at his watch and consulting a time-table he found, however, that by starting immediately he could make a connection at Harrisburg which would enable him to reach Philadelphia in time for his office hours and his lecture the next morning. He accordingly left at once, and, as he told me, reached home in time for breakfast the next day, without having had anything whatever for twenty-four hours except a drink of ice-water on the cars.

Until a few years before his death he usually went up stairs at the hospital at a gait which tried the legs and the wind of the assistants. When operating in private houses heavy persons often had to be lifted from bed to operating-table and back again. Dr. Agnew used to say laughingly that there was no one we could not manage between us, and he continued to do his share, and more than his share, of such work until I positively refused to permit it. Physical labor never unsteadied his hand or affected his operating in the slightest degree, and he was disposed to speak rather disparagingly, so far as his

kindly nature permitted, of those who were thus affected. Among other minor peculiarities of fellow-surgeons which he did not admire was the disposition to invent and employ a multiplicity of instruments. He made some additions to the surgical armamentarium, as I have already mentioned, but he never laid special stress upon them, and he habitually employed the fewest and simplest instruments possible.

As an example of his extraordinary endurance, and also of his physical courage, I may mention the fact that during a cholera epidemic in Philadelphia he has, on a broiling summer night, gone over to the pit in which the unclaimed bodies were placed, and has himself injected as many as fifteen at one time, an amount of labor and a degree of risk to which few men would be willing to subject themselves. His experiences in procuring dissecting material were many and peculiar. He did not hesitate in the case of unknown vagrants or paupers, where no feelings could be lacerated or shock inflicted upon surviving relatives, to step slightly beyond the strict letter of the law in the interests of science, and even has gone out to the Potter's Field at midnight, resurrected a body, placed it in a sack in the seat of his buggy alongside of him, and driven it in to his Chant Street dissecting-room.

He had a keen sense of humor, which never failed to respond to a good story or a jocular remark, and was always thoroughly appreciative of innocent fun of any sort. He could on occasion be very amusing, a quaint vein of harmless sarcasm running through his accounts of his numerous experiences. I have heard him more than once tell of an incident that occurred while he was living at 1611 Chestnut Street. A man, residing in Williamsport, had half swallowed his artificial denture, which became impacted in the œsophagus. He was sent down to Dr. Agnew, who met him at the Colonnade Hotel and extracted the plate. An enterprising reporter who heard of it rang Dr. Agnew's bell at one o'clock the following morning. Mrs. Agnew, who often interposed between her husband and unnecessary demands upon his time and strength, spoke to the visitor from the window. He said that he had learned that

there was a man in town with a set of teeth in his throat, and he wanted to get the particulars. She reported this to Dr. Agnew, and asked what she should say. He said: "Tell him that if he doesn't go away from here promptly there will be another man in town in a few minutes with a set of teeth in his throat."

In his persistent cheerfulness he resembled most of the great surgeons of whose histories we have any accurate record. Larrey enlivened even the retreat from Moscow with his pleasantries; Astley Cooper was the life of his medical club; Abernethy was famous for his dry witticisms, and many other lesser lights were notably humorous.

Much of his success was undoubtedly due to his sympathetic temperament, his native eloquence when discussing any subject on which he felt deeply, and his remarkable self-denial in the pursuit of that science to which his life had been so thoroughly and completely devoted. There are not many instances, apart from those of religious fanaticism, of such utter abandonment of ordinary aims and ambitions, and of such intense concentration of all mental and physical energies upon one idea, as characterized Dr. Agnew during the years when the struggle was hardest and the reward seemed doubtful or distant. While slow to go beyond his self-appointed sphere of thought and action, he was prompt to resent interference from those whose knowledge and research he knew to be superficial and limited; and any suspicion of imposition or falsehood was sufficient to excite in him the most profound contempt. Thoroughly honest in all his convictions, making no effort to appear what he was not, and never influenced by any but the most upright and conscientious motives, he despised all hypocrisy in others, and above all in those who prostituted our profession to what he considered their own personal aggrandizement. He was not, however, Quixotic in his ideas, and his native shrewdness and common sense enabled him to appreciate fully the merits of those men who, like himself, gained both professional and pecuniary success, and combined properly and deservedly scientific with material progress.

He never asked and rarely obtained in individual cases the fees to which his rank in the profession entitled him, although the aggregate of his earnings, on account of his enormous capacity for work, was very large. His small charges arose partly from the habit formed during his earlier days of practice, partly from a dislike for the drudgery of bookkeeping, which resulted, as in the case of the late Dr. Meigs, in many visits being unrecorded and forgotten, but chiefly from his conscientious desire that no sick or ailing person who wanted or needed his opinion should be prevented by lack of means from consulting him. During the later years of his life it became absolutely necessary to limit his practice by raising his fees somewhat, but he was always too modest and had too little of the money-making instinct to place a proper valuation upon his services. I am tempted again to call attention to the parallelism between his habits in this respect and those of Sir Astley Cooper. The language employed by the biographer of the latter might be applied without the change of a word to Dr. Agnew: "There were two or three classes of persons from whom, if he knew their occupation, he always, throughout life, made it a rule not to take a fee, even when offered to him. When this desire not to receive any remuneration for his advice arose out of a belief that the pecuniary resources of his patient were very limited, he had the most happy manner of expressing it to the patient, without wounding his pride, or otherwise offending his feelings."

Both by character and training Dr. Agnew was law-abiding and scrupulously, even rigidly, correct in all the details of his life; but, on the other hand, for those who, less fortunate in temperament or education or surroundings, succumbed to temptations which to him were so ineffective as to be practically non-existent, his sympathy and compassion were never-failing. No one was farther than he from the Puritanical morality which, negatively virtuous and technically Christian, finds the chief solace for its own discomforts in the contemplation of the failings of others, and its greatest pleasure in sour and sarcastic condemnation of their errors.

Not that he could not be tenacious to the last degree of his own opinions when he considered that a principle was involved. He never varied in his belief that the theatres, against which he once wrote a vigorous philippic, were on the whole both a cause and a symptom of moral deterioration in the community, an index of retrogressive civilization. He never approved of the co-education of the sexes, nor of the medical education of women under any circumstances, believing that any possible advantages to them were far more than counterbalanced by what he thought would be the inevitable loss of dignity and delicacy resulting from the association of the sexes under such conditions and from the character of their studies. In accord with the one opinion he denied himself during his whole life the pleasure which he would undoubtedly have derived from seeing the masterpieces of the drama, many of which he was fond of quoting, enacted on the stage. In accord with the other, he resigned a valuable hospital position, and, in spite of strong pressure, refused many lucrative consultations.

He was never an advocate of what is now called the higher education of women in any direction. He admired the domestic virtues, and at one of the last dinners he ever gave remarked that a woman should be taught housekeeping, hygiene, and belles-lettres. After that, he said, the more she knew the worse off she was.

He valued highly the opinions of those whom he considered his peers in the surgical world, but, as a rule, entirely disregarded all criticism of himself or of his doings, whether favorable or the reverse.

In 1882, after the conclusion of Guiteau's trial, Dr. Hays, editor of the *Medical News*, asked Dr. Agnew to prepare a statement of the Garfield case, which should serve as an answer to the unfavorable criticism of its conduct made by Dr. William Hammond and a few notoriety seekers among the laity. Dr. Agnew at first consented, but on reflection decided that it would not be dignified or proper for him personally to notice such attacks, and asked me to write the paper and state that it met with his approval. This I did, and this

was the only notice he took of the bitter public accusations which involved him equally with the other consultants and the medical attendants. A statement he had made to me, which I had previously published, and a letter he had written to Dr. William Hunt, were evidence that he knew from the beginning the surgical impossibility of locating or extracting the missile, and recognized at the same time the probable hopelessness of the case. But his cool judgment and calm superiority to popular clamor and professional pressure prevented those facts from being made public at a time when their promulgation would have wrought serious harm to great national interests. His high sense of honor in all his relations as a consultant was never better illustrated than in the same case. I cannot even now go into details with propriety, but I may venture to say that had he divorced his interests from those of some of the gentlemen who saw the case earlier, he could easily have avoided all responsibility for those portions of the treatment most open to hostile criticism. This, however, he would not permit, preferring to stand by his professional brethren even under fire, and in a case which he knew would be historical.

His refusal to make a charge for the many days and nights which he had spent with the President during nearly three months, involving a loss of weeks from his practice, then probably the largest and most lucrative ever acquired by an American surgeon, and the niggardly behavior of Congress in appropriating a sum which gave him only $5000, a sum less than he should have received, or could properly have charged for one-tenth the service rendered, are also matters of history.

After his retirement from the chair of surgery he gave each year, at my urgent solicitation, backed by that of the class, a clinic at the University Hospital. It was always known in advance, and the amphitheatre was packed from floor to ceiling. At the first sight of his stately figure and silvered head the applause began, as vociferous and deafening as only a medical class can make it, and continued until by a quiet

motion of the hand he indicated his desire for silence. He never lost his hold on the love and respect of the students, and until the day of his death was the most popular member of the University Faculty.

In 1888 the beginning of degenerative changes was manifested by a violent attack of renal colic. He sent for me one morning about four or five o'clock. I found him sitting in a chair in his bedroom, leaning over the back, bathed in a cold perspiration, with a feeble pulse and in an agony of pain. On inquiry, I learned that this had been his condition for some hours, and on asking him why he had not sent for me sooner found that he had not wanted to disturb me, as he knew I had been out late to see a patient for him the evening before. I felt ashamed when I thought how promptly I would have sent for him if I had had a tithe of the pain from which he was suffering.

His first serious breakdown was in 1889, when he was confined to his bed with influenza. I attended him in conjunction with Dr. William Pepper. He made a slow convalescence, interrupted and prolonged by supra-orbital neuralgia and lumbar myalgia.

In 1890 he fell on the marble floor of his hall and violently struck the back of his head. I was never certain whether this was from a sudden vertigo or not, and have thought that it might account for the development of diabetes, which took place later in that year, when he was again invalided under the care of Dr. James Tyson and myself for a period of some weeks; but the autopsy disclosed no cerebral condition which could have been associated with traumatism.

Soon after he began to develop distinct anginose symptoms.

Last spring, before entering the clinic-room for his usual lecture, he told me that he felt excessively nervous, and after the lecture he said that the excitement had given him a great deal of præcordial pain. He spoke once more in public, at the University dinner at Musical Fund Hall, and had the same experience. He said, as we drove down there, that he had determined to give up public speaking, and regretted that he

had undertaken it on this occasion. If I may be forgiven for pushing a favorite idea a little farther, I should like to note that after describing Cooper's final address, his nephew says: "This was the last time he appeared as a public lecturer, for he found that the excitement of the scene and the exertion of delivery increased his tendency to the attacks to which he had lately been liable."

Those who were present at the dinner will remember Dr. Agnew's description of the members of the Faculty under whom he had graduated in 1838. His words were well chosen, and his account of their peculiarities was most interesting; but it was only too evident that he was no longer fit for even the slight strain which this imposed upon him. As I went down stairs to see him into my carriage, which I had kept waiting for him, he looked so badly that I suggested that I had better go home with him; but with a touch of his old spirit he said: "No; I don't need a chaperon; I'm old enough to be trusted out alone at night."

With the exception of a week or two, when he was laid up with a mild recurrence of the influenza, he was at work all the spring, keeping his office hours and attending the almost daily operations which I had the honor of performing on his patients. His interest in surgery never flagged, and not long before his death, after watching for an hour or more a tedious and difficult dissection of the neck for the removal of a growth, he looked at his watch, saw that it was far past his dinner hour, and said: "Well, I'd rather see that any day than eat dinner."

He never thought that he could be quite happy if he were altogether without occupation. In 1879, when he was sixty-one years of age, he expressed his views upon the question of the retirement of men of advanced years from active work, when, as chairman of a dinner given to Dr. S. D. Gross on the completion of his fifty-first year in the profession, he said: "It is, I fear, too commonly thought in these days of mad haste for preferment, place, or power, that men when they have passed threescore-and-ten years should gracefully retire to the

shades of private and inactive life, leaving the field to younger athletes. This is a great mistake. . . . There is something in the grace and dignity of age, its serene complacency of mind, which, when coupled with an affluent wealth of knowledge and rich stores of observation and experience, renders the presence of old men in our midst pillars of strength not only in a profession like our own, but to the community at large; indeed, to the world."

On the 8th of March last year I removed a recurrent sarcoma of the abdominal wall from a lady, a patient of Dr. Agnew's and Dr. Murray Cheston's, who had been originally operated on by Dr. Agnew thirteen years previously. It is for me a memorable operation, as it proved to be the last one at which I was to have the honor and the comfort of Dr. Agnew's presence. The weather was cold and stormy. Before leaving the house he told me that, if he felt no better the next day, he would expect me to operate for him on an epithelioma of the lip in a patient of Dr. E. W. Watson's. Early the following morning I had a note, the last I ever received from him, saying that as he was quite well, and as the operation was so trifling, he would do it himself. I ventured to write him and advise against it, but ineffectually. Dr. Watson tells me that it was only with the greatest difficulty that Dr. Agnew succeeded in removing the little growth, as immediately after his arrival at the patient's house the anginose pains were so severe that he was unable to stand, and he required full doses of brandy and some minutes' rest before and during the operation, which was the last he ever performed. He saw another patient later in the day in consultation, and climbed two flights of stairs to do so. This brought on such a severe attack of cardiac pain that it was with difficulty he reached home. He was then utterly prostrated, and lay in the operating chair in his office for some hours. During this time Dr. DaCosta, who came over at once from his office, saw him, and tells me that he regarded his condition as extremely critical. I visited him an hour or two later in response to an urgent note from Mrs. Agnew, which I found awaiting me at my

house late in the afternoon. He was then cheerful and free from pain, and gave me the above account of his day. With the help of Dr. Edward Martin and Dr. George de Schweinitz I carried him up to his bed, which he never left again.

I am sure I may say that in the days that followed he was well and tenderly cared for. In addition to the loving and watchful attention which his devoted wife and niece bestowed upon him incessantly, day and night, he was almost never without a physician in immediate attendance. Dr. Alfred Wood and Dr. Judson Daland divided the days with me; I spent each night in the house and saw him at short intervals; and the consultants, Drs. DaCosta and Pepper, were assiduous and untiring in their efforts to bring about his recovery. There were dozens of volunteers who would have considered it, as we did, a privilege to evince their love and respect by similar services.

During this last illness he showed the same sweet, uncomplaining, well-balanced disposition that had characterized him all his life. At first, even in the thick of the grave paroxysms of angina pectoris that gave him such distress, his professional judgment remained unimpaired, and the most valuable and practical suggestions received by the attending physicians and consultants came from the patient himself. Later, when diabetes re-developed and the brain began to become clouded, he dozed for most of the time, but still retained his old interest in everything relating to surgery. When, after an absence of an hour or two, I would return to his bedside he would invariably ask as to what had occurred surgically, and the formula, "What's new?" on his lips meant, "What operation have you been doing? what cases have you been seeing?" An operation postponed on account of his illness was remembered and inquired about the day before he died. At about the same time, in conversation with him, mention was made of a mutual friend who had recently undergone a severe operation upon the face. Dr. Agnew was quiet for some minutes, and those about the bed spoke of other matters, but he opened his eyes suddenly and said, "Is the scar well placed?" I at

first thought he was wandering, and made some remark about the effect of the morphine he had taken, but he speedily made clear that he wanted accurate information as to an important detail in the surgery of the case. "Did you take that breast out?" was the greeting on another occasion, when the mind was becoming feeble, and when it was supposed that all outside interests had departed, but the question referred to a specific operation upon a relative of one of his colleagues.

The attacks of angina, which at first were brought on by even a change of position in bed, gradually diminished in frequency and severity, and for some days the outlook was promising, but later diabetes reappeared; he grew dull and drowsy, stupor deepened into coma, and he died on March 22d, surrounded by loving friends and relatives. The announcement of his death was followed by manifestations of grief so widespread and so striking as to make it assume the proportion of a public calamity, and no one who was present at his funeral and saw the stream of persons of all ages and conditions who filed past his coffin could have failed to be impressed by the hold which he had upon the respect and affection of this community.

It is to be noted that the cardiac conditions found at the autopsy were similar to those found in John Hunter and Sir Astley Cooper.

I have had the good fortune to number among my acquaintances, and I think I may say my friends, four whose names could not be omitted from the roll of the great men of this century. Two were devotees of pure science, Agassiz and Leidy; two were surgeons, Agnew and Lister. I have over and over again thought of the remarkable similarity in their personal characteristics, which was so striking that a description of one applies equally to all. Simple and unaffected in their manners, kind and courteous in their treatment of everyone without regard to social conditions; slow to take and even slower to give offence; broad in all their views of life and its complex relations; modest to the point of diffidence where their own merits were concerned; ready to give freely and

earnestly from their overflowing stores to the humblest seeker after knowledge; earnest and sincere without being quarrelsome; good with no shadow of self-righteousness, they seem to me to hold up to poor human nature examples that should stimulate and encourage even the worst or the weakest among us.

The words which my father, whose pride it was to count Leidy and Agnew among his personal friends, used in an obituary memoir of the former, would describe them all: "Appreciation of his rare intellectual gifts was forgotten in admiration of his sincere, sweet-tempered, loving nature. Retiring and unassuming; genial and kindly in spirit and manner, the friend of all, the enemy of none; as approachable as a child, ready at all times and with evident pleasure to give the benefit of his knowledge to all who sought it, his death will be mourned wherever science is valued throughout the earth; but we especially will miss his kindly face, his ready hand, his cordial greeting, and his noble example of industry, integrity, and manly character."

By the death of Dr. Agnew our profession suffered a loss of the greatest magnitude, and one which is, perhaps, altogether irreparable. There remain men equally eminent in some departments of surgery; men of equal ability as public expounders of surgical principles; men whose earnest and self-sacrificing devotion to medicine in its broadest scope cannot for one moment be questioned; but there is probably not one who, combining in himself all these qualities, possesses in addition the skill in observation, the personal magnetism, the singleness of soul, the ceaseless, untiring energy which made Dr. Agnew not only one of the greatest surgeons, but also one of the best citizens of this or any other country. When I come to review finally his record in the many important relations of his life, and remember his unswerving advocacy of all that tended to raise the standard of teaching and of our profession; his resolute support of every progressive movement at the institutions with which he was connected; his unsurpassed ability as a clinical and didactic lecturer, and

his conscientious discharge of the duties of those positions; his remarkable work as a practitioner, operator, and consultant; his genuine love for that work, and his eager desire to acquire any new fact that might benefit his patients; his respect for every honest opinion, even if it differed from his own; his quick and ready sympathy, and his tender treatment of all who claimed his help; his generous support of the feeble, the halting, and the unfortunate in the profession; his gentle courtesy, kindly bearing, and warm friendship which never failed in time of need, I can well believe not only that our profession has suffered an irreparable loss, but that there are many who, like myself, feel that they have sustained a personal bereavement, which time may soften but cannot efface.

It is a consolation, gentlemen, to know that he lived to receive and appreciate the highest rewards that the profession has it in its power to offer. In the poem which our President read to him on the occasion of his jubilee in 1888, he recalled the imaginative promise of Minerva to the lad who a half-century earlier had taken his degree in medicine:

> "But I will take you where the great have gone,
> And I will set your feet in honor's ways;
> Friends I will give, and length of crowded years,
> And crown your manhood with a Nation's praise."

When we mourn his departure from among us, and miss his strong hand and friendly face and wise counsel, it is at least some comfort to know that the honor and the friends and the praise came to him without stint, and that he died in the knowledge that he was the well-beloved, the "Dear Old Man" of the profession.

NOTICE OF THE LIFE AND CHARACTER OF GEORGE CHEYNE SHATTUCK, M.D.,

An Associate Fellow of the College.

By ALFRED STILLÉ, M.D.

[Read June 7, 1893.]

The request made by the College that I should "prepare a minute expressive of the regret of the College at the death of Dr. George C. Shattuck, its Associate Fellow," would have been complied with literally had I not felt that his name alone can stand for very little in the minds of the present generation of the Fellows, and that, therefore, some account of his life and character might perhaps prove interesting, and even useful, to the many whose pilgrimage lies still in greater part before them. And perhaps it may be pardoned to one who feels the sundering of the ties of a friendship he has enjoyed for fifty-seven years, if he desires that others may know the grounds of the honor and esteem in which his friend was held, not only by himself, but by the medical profession of his native city and State, as well as in a sphere far wider but of a different nature.

The "glittering generality" that "all men are born free and equal," if not yet eliminated from political creeds, has never ceased to be a fallacy. Men are not only what their environment makes them, but also what they become through inherited traits and tendencies—physical, moral, and intellectual. At every step the inquirer takes in the realm of history or

biography he encounters many in whom the same dominant defects, talents, vices, and virtues are transmitted from generation to generation as certainly as the physical peculiarities of families. As the popular saying expresses it, "they run in the blood." In New England, where, until quite recently, the race was unmixed, family traits and family pursuits have long been hereditary. Dr Shattuck's paternal grandfather was a physician, and his father, after graduating at Dartmouth in 1806, and at the University of Pennsylvania in 1807, became one of the foremost practitioners of Boston. For three successive years he won the Boylston prize for essays on "Mortification," on "The Anatomy and Physiology of the Skin," and on "The Causes, Diagnosis, and Cure of Biliary Concretions," all of which testify equally to his learning and his clinical knowledge. In 1828 he read before the Massachusetts Medical Society a dissertation on "The Uncertainty of the Healing Art." Like his son, he devoted himself to the laborious duties of an extensive practice, although the possession of an ample fortune absolved him from the need of work; like his son, "he gave free play to his generous feelings," and was a "faithful dispenser," as he said, "of the blessings Providence intrusted to him for the good of others." Like his son, also, he never desisted from the duties of his profession until the symptoms of a dangerous and insidious disease of the heart warned him, at the peril of his life, to retire from the field of his active labors. In the same generous spirit as his son, he of his wealth "contributed largely to the library of Dartmouth College and built an observatory for that institution." He also founded and endowed "the Professorship of Pathological Anatomy" in Harvard University. He was a member of many medical societies, and in 1839 was elected an Associate Fellow of this College. He died in Boston in 1854.

His son and namesake, George Cheyne Shattuck, Jr., was born in Boston, September, 1813. He received his early education at Round Hill, Northampton, Mass., while that school was famous under the government of Cogswell and Bancroft, the former celebrated as a man of learning, and afterward as

librarian of the Astor Library, and the latter reaching the first rank of American historians and statesmen. Dr. Shattuck graduated in the arts at Harvard in 1831, and for a year pursued the study of law, which he gladly forsook for the more congenial and hereditary cultivation of medicine, securing in due course from the Harvard Medical School the degree of M.D., and extending his professional studies at Bowdoin and at Burlington, Vt. In the autumn of that year he crossed the ocean, and in Paris applied himself with exemplary diligence and singular tenacity to the study of medicine, and especially to its clinical branches. For this purpose he frequented the wards of Andral, Chomel, and Louis, who at that time were the most famous representatives of clinical medicine. No doubt his preference for these instructors was determined by the letters to them which he carried from his immediate American predecessors in the Parisian hospitals, including his townsmen, James Jackson, Bowditch, and Holmes. They headed the long list of American students who drank eagerly of the fountains of professional knowledge then and for a long time afterward open to foreigners in Paris. Among them were Dubois, Swett, and Clark, then, or later, of New York; Gerhard, Pennock, and Stewardson, of Philadelphia; Power, of Baltimore; Gaillard, of Charleston, every one of whom held, later on, a professorship in this country, or was distinguished as a teacher or writer. It is probable that between Louis and Shattuck there existed a natural affinity, growing out of the simplicity of their habits, amounting almost to stoicism. The transparent honesty of their characters, their absolute truthfulness, and their agreement in regard to the foundations of scientific truth, formed a band of friendship between them that was never broken while both lived. As an early mark of their mutual regard, Louis intrusted to his American friend his manuscript history of the yellow fever epidemic at Gibraltar, which he had studied as one of a commission sent thither by the French Government in 1828. This valuable memoir Dr. Shattuck translated and published at his own expense. At Louis's suggestion Shattuck made a comparative study of typhus and

typhoid fever, the former in the hospitals of Great Britain and Ireland, and the latter in Paris, and his conclusions confirmed in every point those at which Gerhard and Pennock had arrived by a similar study in the Almshouse Hospital of Philadelphia in 1836. Dr. Shattuck's observations were embodied in a report made by him to the Medical Observation Society of Paris; it forms one of the documents that went to determine the specific differences between the two diseases. The steps by which these differences were established appear to be of sufficient interest to justify their recapitulation here. In 1834, on his return from Europe, Dr. Gerhard was elected a resident physician of the Pennsylvania Hospital, where he instructed a private class, of whom I was one, in medical pathology and diagnosis, and in an especial manner in the clinical history of typhoid fever. In the same year he published a paper showing that our common continued fever was typhoid, and not typhus fever, which he had studied in Edinburgh. In 1836 he was elected an attending physician of the Philadelphia (Almshouse) Hospital, where I served under him. At this time an epidemic of typhus prevailed, and Dr. Gerhard had the good fortune to be able to study it along with typhoid fever. The results of his investigation are contained in the epoch-making history of this epidemic, which he published in the *American Journal of the Medical Sciences*, of February and August, 1837. In the spring and summer of 1838 I visited the fever hospitals of London, Edinburgh, and Dublin, and a prison at Naples, in all of which places I recognized the features of typhus fever that Gerhard had made me familiar with in Philadelphia. In September of the same year I presented to the Société Médicale d'Observation, of Paris, a paper in which I endeavored to demonstrate the diagnosis of this disease from typhoid fever, using for the former Gerhard's and for the latter Louis's detailed description.

Of this paper, Valleix (*Archives gén. de Méd.*, Janv. et Fév., 1839, pp. 69 and 200) remarked (p. 213): "Dans un mémoire inédit de M. le Dr. Stillé, interne de M. Gerhard à l'époque où l'epidémie envahit Philadelphie, mémoire qui a été lu à la Société Médicale d'Observation, et que nous avons sous les yeux, les

deux maladies ont été comparés symptome par symptome, lésion par lésion, et qui, sauf quelques phénomènes qui constituent le mouvement fébrile de toutes les phlegmasies, on peut être sûr de trouver dans l'nn tout le contraire de ce qu'on rencontre dans l'autre." In this article no allusion is made to Dr. Shattuck's investigations, but it would seem that the author must have afterward become acquainted with them, since in his *Guide du Médecin practicien*, 1847, x. 823, Valleix, after citing Gerhard's history of the two diseases, thus proceeds: "Plus tard un observateur habile, M. Shattuck, de Boston, m'ayant communiqué plusienrs observations recueillées à Londres, j'en fis l'analyse" ("Du typh. et la fièvre typh. d'Angleterre," *Arch. gén.*, Janv. et Fév., 1839). And at page 825 we read: "C'est surtout d'après la rélation donnée par M. Gerhard, et d'après l'analyse des faits observés par M. Shattuck qu'il convient de trace la déscription des symptomes."

So Grisolle (*Pathologie interne*, 2éme éd., 1846, p. 65), after describing the diagnosis established by Gerhard, says: "Depuis lui un médecin également distingué, notre ami Shattuck, de Boston, a réceuillé pendant son séjour à Londres des observations precieuses qui ont confirmé les resultats de son compatriote."

Murchison (*Continued Fevers of Great Britain*, 2d ed., 1873, p. 431) also testifies as follows: "In February, 1839, Dr. Shattuck, of Boston, U. S., came over from Paris, where he had already studied enteric fever, and watched some cases at the London Fever Hospital. He wrote an account of thirteen cases, which he communicated to the Medical Society of Observation of Paris. . . . Dr. Shattuck strongly insisted on the existence of two forms in England."

During two winters that I lived in Paris I had every opportunity of observing Shattuck's conscientious devotion and unwearied diligence in acquiring professional knowledge—sacrificing for its sake many of the pleasures in which even the more studious of his fellow-countrymen sought relaxation from the toil of early morning clinics and mid-day special classes, and afternoon revisitation of the wards for the study

of medical specialties—under such teachers as Grisolle, Barth, Valleix, Dépaul, and others. If his daily bread had depended upon his assiduity he could not have been more devoted to the pursuit of knowledge or spent upon it more earnest labor. Among his associates in these engrossing studies few equalled him in his constancy and industry, and fewer acquired knowledge from them in so clear and permanent a shape, or made them as fruitful and helpful in their professional career.

In the spring of 1837 he was one of a party who travelled in the south of France and in Italy, and while he enjoyed the novelty of the natural and social aspects of those countries, and was interested in their historical monuments and in their marvellous productions in the fine arts, he derived a livelier pleasure from the impressions made by the splendor of their churches and their rites and ceremonies, and by the charitable institutions they had founded and maintained. At a much later period of his life he found in this respect a more congenial moral atmosphere in England, and formed there many lasting friendships with eminent men.

After an absence of nearly three years, Dr. Shattuck returned to Boston, and, unlike so many other Americans who studied abroad, he carried home with him some of the glow which they are so apt to find grow cold on returning to their native land. With him, if the enthusiasm was more moderate, so, too, was the disappointment less. He lost no time in making the opening which he did not find. In the absence of any hospital appointment he established a private clinic, or *ambulatorium*, in which he gave instruction to his private students, and thus kept fresh his foreign acquisitions and added to them constantly of his own, while he trained many young men in those pure clinical methods which he considered indispensable, and, indeed, the only true way and safe guide to medical truth. In this he followed the example set by Dr. Gerhard, of this city, who was, I believe, the first to give true clinical instruction in America, in 1835–36, while he was still a resident physician of the Pennsylvania Hospital. From his instruction, as from Dr. Shattuck's, all speculative inquiries were banished;

those teachers, and the school to which they belonged, considering that it was much more important to discover the laws of Nature than to invent reasons for those laws. It would seem as if the logical simplicity of this medical creed must have been singularly attractive to the physicians of Boston, for while among the ignorant or half-educated practitioners of other portions of New England, system-makers have not been wanting, we find that in the long line of eminent physicians of that city there is not one who became addicted to spinning the flimsiest of all theories—medical hypotheses. The methods of the Parisian School of Observation, as formulated by Louis, found in Boston a fitting soil for their culture, and enthusiastic missionaries in the persons of Louis's pupils, including James Jackson, Jr., Holmes, Bowditch, Shattuck, and yet others, who formed there a society which still survives, modelled after the Medical Observation Society of Paris, of which Louis was the founder and perpetual president.

For quite ten years Dr. Shattuck labored earnestly in this narrow but fruitful field, when, in 1849, he began to reap his reward in being elected one of the attending physicians of the Massachusetts General Hospital. For the very unusual period of thirty-six years he continued to discharge the duties of his office with ability and zeal. In what esteem he must have been held is shown by his receiving, in 1855, six years after the clinical appointment, that of Professor of Clinical Medicine, and four years later the chair of Theory and Practice of Medicine, in the Harvard Medical School. This chair he occupied for fifteen years (until 1874), and during the greater part of the time served as Dean of the Medical Faculty. Throughout this period he obtained, as he deserved, great credit for his persistent efforts to widen the scope and improve the methods of teaching, not only within the lines of the official programme, but by his own zealous and extra-official labors. This, too, during a part of the time when some of his colleagues inclined to prolong the placid slumber in which, until then, nearly all the medical schools of the United States had indulged. Happily, he lived to see his alma mater awake

from the lethargy of her self-satisfied optimism, and become the leader of American medical colleges in their march toward loftier ideals and more valuable achievements It was this honorable and distinguished record that caused Dr. Shattuck's name to be included in the list of Associate Fellows whom this College received with impressive ceremonies at its centennial celebration in 1887; and some may remember the characteristically wise and humorous remarks in which he proposed a toast "To the continued health and wealth of the College of Physicians of Philadelphia."

It may not be proper on this occasion, nor in this place, to depict Dr. Shattuck's career in the religious world, to which his time, his means, and his affections were for many years given without stint. To him that world was only a sublime field for the exercise of all the traits of character which he conceived should adorn a medical career. Within its sphere his noble and generous character was admired by everyone, and since his death some of the most eloquent tributes to his memory have proceeded from the clergy and leading laymen of the church to which he belonged. Neither in religion nor in any other sphere were his feelings bounded by sectarian partisanship; for while his own views were definite and stable, his practical relations with other men never suffered on this account, nor was he restrained by it from promoting whatever tended to enlighten and improve and benefit his fellow-men. The famous St. Paul's school at Concord, N. H., was founded by him, and he assisted in establishing a similar one, the Shattuck school at Faribault, in Minnesota. For a number of years he delivered annual lectures on physiology and hygiene to the students of Trinity College, Hartford, Conn., and of St. James College, Md. Thus was he never weary of well-doing, filling and rounding his life with intellectual and moral wealth, and not for himself, but to make others useful and happy.

As Dr. Shattuck gradually withdrew from his public medical duties he became more actively engaged in those which have just been alluded to, yet he did not forget the pensioners on his medical resources, nor did he feel obliged to transfer them

to other hands until several years before his death. Already he had been warned that his heart was beginning to fail, but it presented no urgent symptoms. In March, 1891, he was overturned in his carriage and suffered "severe bruising, one or two lacerated wounds of his arm, and lost part of the last phalanx of the middle finger of his right hand." But for the last year of his life he had good use of the limb. When the direct effects of the accident had in a great degree disappeared, he found it very difficult to resign himself to the inaction which had become necessary. For the last week or two of his life his respiration was difficult, and there was a good deal of discomfort, and sometimes suffering, from the interference with the heart's action. "He bore everything with patience and cheerfulness. His attendants became greatly attached to him, and he was very thoughtful about giving them trouble." His mind remained clear almost to the last. The explanation of his symptoms was found in an aneurism of the left ventricle, with hypertrophy and dilatation of the heart, ossification of the aortic valves, and sclerosis of the coronary arteries.

Dr. Shattuck's death was felt almost as a public calamity in Boston, where his benefactions had been so munificent and his benevolence so widespread. The newspapers were outspoken in their eulogies. They said of him that "perhaps no one in Boston had done more good to a greater number of people;" that "his liberality of opinion was equal to his loyalty, and religious and political creeds were no barriers to his friendship;" and that "his kindness, courtesy, and helpfulness had the quality of paternal benevolence. He did not give of goods alone, but of tender ministration, of kind offices, of whatever might be appropriate in each case. His bounty was spontaneous, efficient, and without the slightest pride or ostentation."

Of all men who were his equals in social rank he was the least pretentious, or, rather, he was the most humble, of any I have ever known; the most charitable in his judgments, and the most generous in his deeds. Nothing sour, or solemn, or

dismal tainted his manner or his character. To him religious faith brightened the world and infused a cheering and ennobling spirit; it gave a warmer tint to the joyous humor he so largely inherited, and which made him so delightful a companion. He had seen many men and countries, and had been brought into close contact and even cordial friendship with eminent persons at home and abroad, and he was quick to seize their salient traits of character and manner and reproduce them in his conversation. If from out of the bundle of his virtues I were to select the one that overtopped all the rest, I should call it TRUTH!—truth in all that he thought and felt, in all that he said or did, in all that he was; and that rounded his character and made him, as far as human nature admitted, a perfect man.

As not a few among my hearers know, he has left behind him two sons, who bid fair in perpetuating his name to adorn no less than their father the calling that he honored and so faithfully served.

Venturing to lay before the College this tribute to the memory of our late Associate, I beg leave to add a minute, such as I was specifically appointed to prepare, as follows:

By Dr. Shattuck's earnest devotion to the duties and interests of the profession for which he felt an hereditary attachment, and which he illustrated by a long career of fruitful teaching and practice, and by the uprightness, unselfishness, and simplicity of his life, and his genial and benevolent disposition, he exerted a beneficent influence while he lived, and has left behind him the memory of a character worthy of admiration, and which should serve as an example and encouragement to all who aspire to be held in honor and affectionate remembrance.

REMARKS.

DR. WILLIAM OSLER: I wish to add a word and to ask one or two questions, particularly with reference to the part Dr. Shattuck played in Paris in the elucidation of the problems of typhoid and typhus fever. I should like to know whether or not Dr. Shattuck's lectures were published. I do not

remember to have seen them. I know that Dr. Stillé's paper before the same society was not published, although I have had the opportunity of examining it in the manuscript. It is a great satisfaction to feel that the work which was done in Philadelphia by Dr. Gerhard, and in Paris by Dr. Shattock, and presented before the Paris Society for Medical Observation by Drs. Stillé and Shattuck, had much to do with modifying Louis's views as to the identity of the two affections.

Another point that has often struck me as very interesting is the remarkable influence which Louis's students have had upon the profession in this country. We, of this generation, are apt to forget that the men who really brought the scientific instinct and the desire for careful scientific study to this country were the men of whom Dr. Stillé has spoken, and of these it is sad to think that Dr. Stillé and Dr. Holmes alone remain. These are the men who must be held in honorable remembrance by the profession, not only for the work which they have done, but for the example which they have given.

Dr. Stillé: I am not quite certain whether or not Dr. Shattuck's observations were published. That question occurred to myself in preparing the paper, but to pursue it I thought would lead me into greater detail than was fitting. I might have said briefly that he, like myself, presented to the Paris Society for Medical Observation a memoir founded upon his studies in England, particularly in the Fever Hospital of London and in the Royal Infirmary of Edinburgh, and also in Dublin. I cannot remember which of us preceded the other, but I think that Dr. Shattuck was in advance of me in the London wards. Whether he presented to the Society his notes on the resemblances and differences of typhoid and typhus fever before or after my own, I am not sure. It seems to me, however, that they were published, if not in full, in part, in the *Medical Examiner* about 1843.

ANNUAL ADDRESS OF THE PRESIDENT.

By S. WEIR MITCHELL, M.D.

[Delivered December 6, 1893.]

Fellows of the College of Physicians: Perhaps you are fortunate in the fact that your President finds little to state in the annual address required by your by-law. There are certain needs which may find mention in the reports of your committees, but which by their importance justify a word from the Chair. One is the ever-increasing want of a large sum to be expended in a new roof. Before long, and perhaps at some time quite abruptly, this need may grow to be instant and imperative. The Council should, I think, consider this matter, as also the necessity for added stack room in the library.

I have authorized the formation of Sections under the somewhat stringent conditions made by the College. These new bodies are still in the experimental stage. You now have Sections of Ophthalmic Medicine, Orthopædics, Otology, and Laryngology. Others will soon be asked for. The Sections have not increased too rapidly. A report of their work will reach you through your Secretary. In this connection I ask that the Council consider the absolute need to give the Secretary clerical aid.

During the past year the College has been efficient in public duties, has most satisfactorily influenced the mode of paving our lanes and alleys, has been felt in the legislation anticipative of cholera, and has been represented through the Fellows appointed by me in accordance with law to represent it on the State Board of Quarantine. The able Cholera Commission,

composed of your Fellows, was, as you will learn, active during the summer, and should be continued through 1894, with power to fill vacancies.

I feel it to be my duty as your President to call your attention to the wisdom of some protest against including all tubercular disease in the list of maladies officially recognized as contagious. Even if we admit it as of this nature, it would be clear that no sufficient good could result from the extreme measure in question. Is it proposed that the Board of Health shall interfere actively in any case of consumption, and how? Is it to take cognizance of all tubercle or of tuberculous discharge from the ear or from a gland? Where is the line to be drawn, and what protective measures are to be taken by the Board of Health? The futility of the contemplated measure is plain; it invites a form of deceit hardly to be blamed. It is so temptingly easy to consider the bronchitis of consumption as the true enemy and elect thus to label the disease rather than to return it as tubercle to a too inquisitive Board. This is not the place fully to argue this case. It may suffice to add that our real duty at present is personally and through Boards of Health to educate opinion, and to direct and teach precautions as to antiseptic means and habits. It is but too easy to create around the consumptive a moral atmosphere of terror. For the nurse or relative this would often be destructive of duty, and for the sick a horrible addition to the miseries of a disease which may last for years. Already this mischief is at work in other forms, and already within two years the monomania of germs and bacilli has won a place with electricity among the means of torment acquired by the educated ingenuity of the insane. I trust that you will protest with energy against any present effort to class tubercle with smallpox, and that a suitable committee, with power to act, be instructed to promptly place our views before the Board of Health. One of your Fellows will this evening present this urgent matter in a shape to permit of immediate action.

Of late years the College has enlarged its fellowship with, I

think, reasonable care as to the quality of the additions. There are those who think its doors should be more widely opened. I am not of this opinion, and I trust that always scrupulous attention will be given to see that we admit only such as are in all ways worthy. I should much prefer that no man under thirty be elected. Nowadays men come into the profession later than was wont to be the case, and more and more it is difficult in a great city to know of them fully all that it is desirable to know. But at thirty a man has become, as a rule, well enough known and we are less likely to make mistakes.

There has been of late too much outside canvassing for the Fellowships. It is well that gentlemen not in our ancient body should signify their desire to be of it. Indeed this was once a formal rule. It is not so now, and direct personal efforts to secure the votes of Councillors ought to insure reproof as being remote from the decent traditions of our body.

I desire to call your attention to the fact that over and over during the past year there have been many infractions of your rule which obliges a Fellow to name to the Secretary guests invited to be present at the meetings of the College. On a recent interesting occasion there were at least twenty or thirty such persons present, of both sexes, entirely unvouched for by a single Fellow. On any future occasion, after the warning given, the Chair will feel obliged to enforce the rule of the College and to ask an unaccredited guest to withdraw.

In nominating Fellows for standing committees, I pray you to recall the rule which you have made, that no man may by election serve on two such committees. Also I think it well to ask that some thought be given to the constitution of committees, and that you do not encourage the democratic stupidity of large, frequent, or complete changes, remembering that it takes time to become familiar with the work required of them.

At present the President appoints the Committees on Finance, Entertainment, the Jenks prize, and the Alvarenga prize. The Committees on Publication, Library, Museum, and Directory for Nurses are elective. I am inclined to believe

that, as the College enlarges, the day will come when you will make it the President's duty to appoint all committees with the advice and consent of your Senate, the Council. In that case you will probably desire that one or more members of the committee be displaced each year, or every second year. I am not secure enough as to the present wisdom of this change, to ask to have it referred. I put it forth as an earnest indication of my hope that in your nominations you will be thoughtful of the need to elect Fellows who have such acquirements as may fit them for their duties.

I have observed that our *Transactions* are limited to our purely scientific matters, and do not present to the world other evidence of our growth in usefulness. The *Transactions* should carry always a full statement of the condition and growth of our great library, as is common in all such society proceedings elsewhere. Some authority may be required, but I think there is only need for the Library Committee to send to the Publication Committee what it considers desirable to print.

You have learned, with such regret as I most deeply share, that your Treasurer, Dr. Charles S. Wurtz, is unwilling to act longer in this difficult office. For seventeen years he has served us with fidelity, accuracy, and thoughtful care as to all of our moneyed affairs; others might have been found to do as well his mere commercial duties, but the constant good humor, the sweetness of conduct under all circumstances, the charm of unfaltering serenity of manners which made this official creditor so pleasant to all men—these I think we shall not easily match.

Four Fellows have died during the official year ending December, 1893.

Dr. E. B. Shapleigh, born in 1823, a Fellow since 1868, and long an active practitioner, was at one time physician to the Coroner. He closed a busy and useful life in December, 1892.

Dr. John C. Hall, born in 1843, of the Fellowship since 1879, succeeded Dr. Worthington as physician to the Friends' Hospital for the Insane in 1877, and continued to hold this post until his death in the present year. Dr. Hall was an active-

minded physician with a large desire to aid progress in his branch of medicine. Among those who are known by the most unfortunate name of Superintendents of Hospitals for the Insane, Dr. Hall stood high. He shared with a very few professional alienists in the belief that the present mode of conducting hospitals for the alien in mind is not the best attainable. Despite the traditions and difficulties of his anomalous position, Dr. Hall did much to improve his hospital, and it would be hard to conceive of a more wisely righteous life or of more single-minded devotion to the interests of his patients.

Dr. Louis K. Baldwin, born in December, 1836, a Fellow in 1876, was in service as acting executive officer of Satterlee Hospital during the war. In active practice till 1890, he thenceforward devoted himself wholly to life insurance and died in the present year.

I record with strong personal feeling the recent death of Dr. John M. Keating, a Fellow from 1877, which ended a career full of brilliant promise over and over disturbed by failures of health. Nevertheless, Dr. John Keating had become exceedingly acceptable as an authority on pædiatrics, and had he had physical vigor to match his intellect and his ambitions, must surely have won a place in the highest rank. He died suddenly in the mid life of a broken career. I am sure there are none here who do not deeply feel for his father, Dr. William V. Keating, one of our older Fellows, in his hour of loss.

Dr. John Keating brought to our ranks a certain winning pleasantness of nature in union with unusual learning, many accomplishments, and traditions such as will always, I trust, be grateful to this College. As a scholar, a physician, and a gentleman, the death of this Fellow removes an interesting and distinct personality. My own relations with him were those of friendship, and you will pardon me if I use this time and place to make this too brief record of my own deep regret at his early loss.

One Fellow, Dr. Rex, has resigned. Two have forfeited fellowship because of non-payment of dues.

Our Associate Fellow, Dr. George C. Shattuck, also died in

1893. Of this estimable illustration of the best qualities of man and physician I leave his friend, our ex-President, Prof. Stillé, to speak in the minute he has been asked to prepare.

I desire to congratulate the Fellows upon another year of wholesome progress, and to remind them that our importance as truly representing the profession is to be sustained by aggressive interference in public affairs involving health.

It gives me pleasure to present to the College, in the name of Mrs. Edward Shippen Willing, a portrait of her father, the distinguished surgeon, John Rhea Barton. It forms an interesting addition to our growing gallery of portraits of medical worthies.

ADDITIONS TO THE MÜTTER MUSEUM.

Eight Photographs of Acromegalia. Presented by Dr. S. Solis-Cohen.

Anomalous Circle of Willis. Presented by Dr. J. H. Lloyd.

Photogravure of Prof. Billroth's Surgical Clinic. Presented by Dr. R. J. Dunglison.

Anencephalous Monster. Presented by Dr. Wm. Goodell.

A Fœtus of Six Weeks. Presented by Dr. A. B. Hirst.

Nine Photomicrographs of Pathological Conditions of the Eye. Presented by Dr. G. E. de Schweinitz.

Intra capsular Fracture of the Femur. Presented by Dr. T. S. K. Morton.

Painting of Syphiloderm. Presented by Dr. A. Van Harlingen.

Epithelioma of the Hand. Presented by Dr. John H. Packard.

Photographs of Sections of Syringomyelia. Presented by Dr. James Hendrie Lloyd.

Eye showing Posterior Staphyloma. Presented by Dr. Chas Shaffner.

Ungual Exostosis of the Big Toe. Presented by Dr. G. G. Davis.

Spectacles used by Esquimaux as a Protection against Snow-blindness. Presented by Dr. G. E. de Schweinitz.

Cast and Photograph of a Case of Congenital Hypertrophy of the Fingers. Presented by Dr. T. S. K. Morton.

Photograph of a Case of Amputation of Both Thighs. Presented by Dr. Isaac Massey, West Chester.

Cancer of the Stomach. Presented by Dr. D. J. M. Miller.

Miliary Tuberculosis of the Lungs, etc. Presented by Dr. S. Solis-Cohen.

Fibroid Tumor of the Uterus. Presented by Dr. Stansbury Sutton.

Ovarian Cyst. Presented by Dr. J. Ewing Mears.

Fibroma of the Labium; weight five and a half pounds. Presented by Dr. Stansbury Sutton.

Carcinoma of Kidney. Presented by Dr. Stansbury Sutton.

Hydronephrosis. Presented by Dr. Stansbury Sutton.

Exostosis of the Femur. Presented by Dr. Guy Hinsdale.

Fracture of the Fibula. Presented by Dr. Guy Hinsdale.

Fracture of the Ulna. Presented by Dr. Guy Hinsdale.

Dermoid Cyst. Presented by Dr. Joseph Price.

Uterine Fibroid. Presented by Dr. J. Ewing Mears.

Uterine Fibroid *in situ*. Presented by Dr. Joseph Price.

Model of the Head. Presented by Dr. J. Ewing Mears.

Two Biliary Calculi. Presented by Dr. John H. Packard.

Spindle-cell Sarcoma of the Choroid. Presented by Dr. C. A. Oliver.

Sarcoma of the Choroid. Presented by Dr. C. A. Oliver.

Specimens illustrating Gastro enterostomy. Presented by Dr. John B. Roberts.

Dermoid Cyst. Presented by Dr. J. M. Baldy.

Cancer of the Rectum. Presented by Dr. J. M. Baldy.

Tubal Pregnancy and Tubal Abortion. Presented by Dr. J. M. Baldy.

A Collection of Uterine Fibroids and Pus Tubes. Presented by Dr. J. M. Baldy.

Microscopic Section of Tuberculous Iritis. Presented by Dr. C. A. Oliver.

PIPERAZIN IN THE TREATMENT OF STONE IN THE KIDNEY: REPORT OF CASES.

By DAVID D. STEWART, M.D.,
LECTURER ON CLINICAL MEDICINE IN THE JEFFERSON MEDICAL COLLEGE OF PHILADELPHIA.

[Read January 4, 1893.]

Piperazin, as is perhaps now generally known, is one of the more recent coal-tar derivatives. It is chemically diethylene-diamine, a piperadin in which one CH_2 group is replaced by an amide. It is freely soluble in water, and in cold aqueous solution will dissolve twelve times as much uric acid as will lithium carbonate. Its urate, which is said to be always a neutral salt, is seven times more soluble in water than is the corresponding salt of lithium, the most soluble of the metallic urates. Piperazin is a stable compound, apparently not undergoing decomposition in the organism. It is readily excreted by the kidneys, and may be detected in the urine by appropriate tests in a few hours after a single dose. The knowledge of the latter of the foregoing facts has led naturally to a wide clinical trial of piperazin in the uric acid condition, especially in gout and in uro-lithiasis, with a pretty general unanimity of opinion as to its value in uric acid gravel and calculi, although curious contradictory results are reported as to its influence upon the quality and quantity of urine. It was with a view of testing its action in cases of uro-lithiasis, and concurrently to ascertain for myself its influence upon nitrogenous excretion in the urine, that I began its trial in a number of cases of uric acid diathesis, of which I shall now only report several of suspected renal calculus in which a careful clinical

examination of the remedy over quite a lengthy period was made, with accompanying thorough microscopic and chemical examinations of the urine.

It goes without saying, from what is known of the action of piperazin, that, of all conditions, uric acid gravel and stone is the one most likely to receive substantial benefit from its use. Despite the affinity of piperazin for uric acid, and the extreme solubility of its urate, little can be expected from it in uratic chronic multiple arthritic enlargement, at least in the doses generally administered—15 grains daily. In much larger amounts, such as a drachm or two daily, exhibited over a continuous time, more promising results may be anticipated; whether, however, such doses will be tolerated without injury is yet to be determined. The present unfortunate high price of the drug prevents the solution of this question.

The first of these cases of uro-lithiasis is that of Miss L. F.,[1] aged twenty-eight years, who came under my observation February 12, 1891, with a history of an almost continuous ache in the right loin for seven to ten months. Paroxysms of pain at times occurred, shooting toward the bladder. She had been passing gravel more or less continuously for several years. There were anorexia, constipation, bad taste, leucorrhœa, painful menstruation, and severe headache occurring about twice weekly. She was in a melancholic condition; was persistently low-spirited, the latter, doubtless, being induced somewhat by the recent death of her favorite cousin from a somewhat similar ailment. Her urine was then noted to be highly acid, of a specific gravity of 1028, and to contain neither albumin nor sugar. Microscopically, there were numerous crystals of ammonium urate, large excess of free uric acid, and microscopic (good-sized) calculi of both ammonium urate and uric acid. There were also amorphous urates, a few pus cells, red blood-corpuscles, and epithelium from the kidney's pelvis and from the bladder. There was then present deep-seated tenderness in both flanks, but no palpable tumor. She was placed upon large doses nightly of nascent potassium citrate, was given hot water freely before breakfast, and was directed to take liberally of an alkaline mineral water through the day, and to live in the open air.

From the date of the first visit she was under observation quite constantly, with the exception of an occasional interval of about two months, until

[1] A cousin of the patient upon whom a nephrotomy for calculus pyelo-nephrosis was done by Professor Keen, and reported by us in the Therapeutic Gazette, January, 1892. These two young women strikingly resembled each other in appearance, disposition, and temperament.

piperazin was begun in January, 1892. In the early part of this period her general condition improved somewhat under remedies directed to assisting the impaired digestion and the use of arsenic after meals. The vegetable salts of potassium were given for a number of months in very full doses, intermittently. A lithia water was also taken freely. Potash could not be continued for any length of time, from the almost invariable gastric derangement it occasioned. During this period frequent chemical and microscopic examinations of the urine were made. The result of these was similar to that recorded above. Pus- and red-cells were never present in large amount, nor could pus even be detected by Donne's test. Crystals of uric acid and urates were usually present in great excess, and occasionally calcium oxalate abounded. Cells from the kidney pelvis were common, and on several occasions hyaline and granular casts were seen. The daily quantity of urine was subnormal—about one and one-half pints, and often but seventeen to eighteen ounces—unless considerable doses of potash or large quantities of fluids were taken. The urea was always low, on several occasions averaging between 142 and 216 grains. Attacks of gravel were very frequent, but were usually dissipated by potash, to promptly return on its discontinuance, despite care in diet and attention to general hygiene. For this reason, and because of the disorder of digestion full doses of the alkalies occasioned, she, after a few months, abandoned their use, and then symptoms of stone in the kidney became more pronounced. There was constant severe pain in the loin, with increased tenderness on pressure, and growing fulness, dulness, and resistance in the flank. Professor Keen at this time saw her with me, and, agreeing as to the probable existence of stone, advised a nephrotomy. To this, consent could not be obtained. I now lost sight of the case for several weeks. During this time she took a great deal of lithia water, but no drugs.

Early in January, 1892, when I again saw her, the symptoms were those of calculus hydro-nephrosis. She was passing less than two pints of urine daily, and often scarcely a pint. There was prominence, tenderness, and dulness in the left hypochondriac and iliac regions, the dulness extending toward and near the umbilicus, and, posteriorly, to the usual situation of the kidney. I now began a brief trial of two drugs—diuretin and piperazin—a barren result with which, I informed her, would necessitate immediate operative interference. Diuretin was first prescribed, principally because she was then suffering greatly from migranous headache, which, it was thought, diuretin would relieve. Little was expected from either it or piperazin on the kidney condition. About a drachm daily of the former was taken for a week or ten days, totally without effect on the urine. Piperazin was now ordered in doses of fifteen grains daily. The issue of the use of this remedy seemed quite extraordinary, so promptly and decidedly did benefit accrue. The urine, which had been unaffected in amount by diuretin, after taking piperazin for three or four days, underwent a most decided increase; from but a pint daily it rapidly rose to four pints.

When seen on February 27, 1892, she had been taking piperazin for over two weeks. There had been voided in the preceding nine hours five pints, a much greater quantity than she is aware passing before or since.[1] The loin pain was much diminished, and the fulness and dulness occupied a much less area. Her general condition in the two and a half weeks had also undergone a striking improvement, noticeable to all. Appetite and digestion were asserted to be better than for several years—the bad taste to have disappeared; in short, she looked better and felt generally improved, an amelioration, doubtless, largely of psychical origin, contributed to by the feeling that her condition was no longer one of desperation.

No minute examinations of urine were possible immediately preceding the administration of piperazin, her condition not admitting of delay in its use; nor was any attempted directly after she was seen on this occasion, it being intended, after the condition had ameliorated sufficiently to allow discontinuance of the drug for a time, to undertake systematic examinations. To my regret, also, it was impossible to keep a continuous record of the daily amount of urine passed from the time piperazin was first begun; this was, however, done subsequently, on starting anew with the remedy, after its temporary withdrawal.

From February 12th to April 2d (the last, one of the dates of temporary discontinuance) the daily dose was from 12 to 20 grains, usually the latter. The daily quantity of urine was always fair and often large. She was careful, by direction, to take the same amount of fluid daily, averaging about 1½ pints, not including that in the food eaten.

On March 10th the amount passed was 65½ fluidounces; specific gravity, 1010; faintly acid; urea, 300 grains.

March 11th to 15th, the amounts were 61, 70, 59½, and 70 fluidounces.

March 16th, 50 fluidounces, faintly acid; specific gravity, 1020; urea, 500 grains; uric acid, 17 grains.[2]

March 17th to 23d, the amounts were 62, 47, 63, 64, 62, 54 fluidounces.

March 30th and 31st, 51 and 53 fluidounces.

Piperazin was discontinued from April 2d to 13th. During the period of seven weeks in which the drug was taken, improvement continued, though not so markedly as at first. Pain, or a sensation of soreness in the loin, persisted; much slighter, however, than formerly, and little tenderness was induced by pressure over the area of fulness and dulness in the lumbar region. This last was now present in moderate degree only.

The subjoined table gives the consecutive daily specific gravity and quan-

[1] The passage of this large amount, under the conditions narrated, can only be explained by the giving way of a hydro-nephrosis.

[2] Within these dates other urea examinations were made, but as the urine was partly decomposed, the results were considered untrustworthy, so are not here recorded. A number of other uric acid estimations were also made, but by a method the technique of which was afterward proved faulty, so that no account is here taken of these.

tity of urine, and also the variations in its most important nitrogenous constituents, estimated from mixed twenty-four hour specimens, prior to and following the administration of piperazin.

In all estimations in this paper the acidity, urea, and uric acid were obtained by the following methods: The acidity was estimated by titration with a $\frac{N}{10}$ solution of NaHO; titrations were made as early as possible after receiving the specimens, which were usually sent promptly after mixing at the expiration of each twenty-four hours. Urea was estimated by the hypobromite process (bromine itself being always used) with the apparatus of Parke, Davis & Co. Uric acid was estimated by the now well-known and reliable method of Haycraft; as with the urea estimations, a second of uric acid was often made to confirm the first.*

CASE I.—MISS L. F.

Date.	Daily amount of urine in fluid-ounces.	Specific gravity of mixed 24-hour specimen.	Degree of acidity calculated on 100 c.c. of urine.	Daily elimination of urea in grains.	Daily elimination of uric acid in grains.	Daily dose of piperazin.
April 8	47	1016	22	307	13	None for 6 days.
" 9	50	1015	20	300	11½	" " 7 "
" 11	58	1016	28	450	15	" " 9 "
" 12	42½	1010	15	250	8	" " 10 "
" 13	52	1013	18	331	12	" " 11 "
" 14	63	.....	...	....	...	18 grains of piperazin daily.
" 15	75	.....	...	....	...	18 " " "
" 16	62	.....	...	....	....	18 " " " [1]
" 24	50	1013	12	273	15 2/5	No piperazin for 7 days.
" 25	51	1015	20	273	14 2/3	" " " 8 "
" 26	46½	1011	30	245	11 1/3	" " " 9 "
" 27	56	1012	21	316	15 2/5	" " " 10 "
" 28	43½	1020	30	196	15 3/5	" " " 11 "
" 29	60	1012	14	330	14 2/3	18 grains piperazin taken.
" 30	55	1012	12	420	13 4/5	23 " " "
May 1	50	1020	20	386	15 2/5	33 " " "
" 2	55	1012	20	423	13 4/5	33 " " "
" 3	44	.....	...	....	...	23 " " " [2]
" 5	60	1012	10	440	19 4/10	23 " " "
" 6	44	1020	10	400	20	23 " " "
" 7	53	1016	22	390	11¾	20 " " "
" 8	51	1019	30	280	19	20 " " "
" 9	47	1013	20	342	14½	20 " " "

[1] Through a misunderstanding specimens were not sent on the 14th, 15th, and 16th, so that no estimations could be made. Piperazin was, therefore, stopped until April 28th, that consecutive daily examinations might be again made immediately before taking and while on the drug.

[2] Specimen of urine not sent.

Several microscopic examinations were made between April 8th and May 9th. There were an abundance of amorphous urates, free uric acid, ammonium

* No account of the daily elimination of total urinary solids is here appended, as these may be readily approximately estimated by multiplying the last two figures of the specific gravity of the urine by 2.33 (Christison's formula) or by 2 (Trapp's). This equals the percentage in grammes, calculated on 1000 c.c.

urate and crystals, large quantity of granular débris, and epithelium from the kidney and bladder.

The urine was not examined from this on. An estimation of the amount passed in the twenty-four hours, however, was still frequently made. From May 12th to 24th the daily quantities were, in fluidounces, 47, 44, 36½, 55½, 49½, 56½, 44, 43, 42, 48, 53, 50, 50. During the second half of June and the early part of July similar counts were made, which it is needless to produce here, since they deviated very little from the foregoing, like them being quite normal.

Piperazin was continued in daily quantities of 20 grains until August 1st. It was then withheld, it being concluded that the patient had entirely recovered. Four weeks succeeding its discontinuance the urea was measured for four consecutive days. The amounts were, in fluidounces, 50, 52, 48, 47. All loin pain and discomfort to manipulation in the renal region had completely disappeared when the patient was examined on June 3d, and had not since reappeared when last seen on October 1, 1892, though she was leading a most sedentary life and taking little or no exercise in the open air. When the abdomen was last thoroughly examined, in June, resistance and dulness were still present in a limited portion of the situation previously occupied by the hydro-nephrosis; but no tenderness, even on deep pressure, could be elicited in this region. No gravel has been noticed in the urine at any time from the date of commencement of piperazin, though looked for by the patient. Prior to taking piperazin, the freshly-voided urine almost constantly contained it, but in less amount than when the patient first came under observation. No untoward results occurred from the long-continued employment of piperazin. Menstruation during several monthly periods was profuse and more frequent than had been customary. What effect piperazin had in the production of this I do not know. Appetite and digestion seemed to be better while piperazin was taken. Headache, of which she has always been a sufferer—probably largely due to eye-strain—continues as before.

The second case is one of striking interest, because of the long-continuance of symptoms of stone in the kidney, and the fact that lack of certain common indications of that affection prevented its recognition, and that of an accompanying pronounced pyelitis, by a considerable number of practitioners consulted at various times.

The patient came under observation accidentally. My attention was incidentally called to him as he lay in a paroxysm of colic, in the out-patient ward of the Jefferson Hospital. It appeared that he had been in the habit for some time of going

to the resident about twice weekly for hypodermic injections of morphine, because of recurring attacks of severe pain in the right lumbar region, which came on suddenly, incapacitating him for work, and which were ultimately relieved by rest and full doses of morphine. At first I was inclined to the opinion that the man was a malingerer; but a careful inquiry into his history, and an examination of the urine, which I had him send me on the day in which I first saw him, caused me to take a different view of the case. The following are the chief points in the case:

A. L., aged thirty-eight years; clerk. First seen December 14, 1891. For seven years he had had, at intervals of four days to two weeks, attacks of severe pain in the right loin, occurring suddenly, and lasting from twelve hours to one or two days. Occasionally an attack would continue for almost a week, during which pain would be never absent, though not constantly severe. The initial attack occurred while serving as a United States army private. He was then on the plains, engaged in digging holes for the erection of telegraph-poles. The pain was always deep-seated in the loin. He could not recall it ever shooting toward the bladder or into the testicle, nor was frequent urination a symptom. During and especially succeeding the attacks he had noticed, particularly the past year or so, that the urine was of an unnatural dark-red hue. This he had not recognized as blood, though it undoubtedly was. Vomiting was not an infrequent concomitant of severe attacks. No access of pain could be elicited by manipulation in the renal region during the attacks. There was absolute freedom from pain in the intervals, and repeated examinations of the abdomen revealed no abnormity of any of the viscera.

He was a robust fellow, with good general health apart from these attacks. The habits were temperate. Gravel had never been noticed in his urine, though it might have been present preceding the appearance of the trouble, or even succeeding its onset, as it had not been looked for. He had consulted a number of physicians, who had treated him principally for gastric and intestinal indigestion. One or two had suggested nephralgia. The operation of stretching the ilio-hypogastric and ilio-inguinal nerves had been undertaken by a surgeon a year or so before with a view of thus relieving the attacks. Stone was then suspected, it being asserted that, were it present, the symptoms suggested a new phase of that affection. It is said that the kidney was felt during the operation, and no stone detected. Despite this testimony, I believed the case in all probability one of renal calculus, and an examination of the urine during and following an attack strengthened this opinion. It was of very high color, and, after a day's standing, deposited a dark-red sediment to the extent of one-half the bottle in which it was con-

tained. Microscopically the sediment consisted entirely of blood corpuscles and pus cells. Portions taken from the upper layer had the appearance of a drop of leucocytic blood. The lowest stratum consisted almost entirely of pus-corpuscles. A specimen passed two days after the attack was acid, of light-amber color, specific gravity 1020, and contained $\frac{1}{15}$ per cent. of albumin (Esbach's method). After some hours' standing a sediment was precipitated, which, microscopically, consisted solely of pus cells containing entangled calcium oxalate and uric acid crystals, the last in less amount than the lime oxalate. Sufficient pus was present on decanting the urine to gelatinize the deposit with liquor potassæ, a response always obtained when this test was similarly applied to a number of subsequent specimens.

I represented to the patient that he had beyond doubt a suppurating kidney, which was in all probability due to a stone lodged therein, earnestly advising him to submit to a second operation. But to this advice he would not listen because of his previous experience in this direction.

Because of its unusual interest, the case was kept under observation that a more extended study of his condition and that of the urine could be made. It was thought that he would ultimately submit to an exploratory nephrotomy, and that thus the diagnosis could be confirmed. Little was expected from a resort to drugs because of the pronounced pyelitis complicating the condition.

I made, in all, thirty-two chemical and eighteen microscopic examinations of the urine. On a number of occasions two or three specimens passed at the time of an attack, at its onset, height, and termination, were examined microscopically. Pus was always present in some amount, except on a single occasion. Blood also could be noted always macroscopically and microscopically at its height; never, however, several days after its entire cessation. Epithelium from the kidney pelvis was frequently found, and on three occasions hyaline and epithelial casts were noticed. The urine was always acid when freshly voided; it usually was quite fetid, the latter especially after standing a short time. The mixed twenty-four-hour specimens were turbid, and could not be cleared by heat and filtration. Calcium oxalate crystals were nearly always present in large amount, but occasionally uric acid and urates were in preponderance. Albumin constantly existed in small amount, $\frac{1}{10}$ to $\frac{1}{20}$ per cent., evidently of pyuric origin.

On several occasions the quantity of urine passed during an attack was diminished, thought it was normal in amount at the termination, leading to the supposition that the ureter of the affected kidney might be temporarily closed.

On May 8th, in a mixed specimen of the total twenty-four-hour urine (23 fluidounces) passed at the height of an attack, there was, in three slides examined, but one pus corpuscle seen in one slide, three in a second slide, and none in a third, with no blood disks. There were amorphous urates and uric acid crystals. The examined portion was taken from different layers of

the sediment, which, in this single specimen, was evidently due to amorphous urates and not to pus. This would indicate temporary obstruction of the ureter of the diseased kidney during this attack; for, also, immediately following it (May 9th) blood and pus were present in some quantity. A daily estimate of the amount of urine passed was made, by my direction, for several months. But symptoms of closure of the ureter occurred with no frequency or constancy of relation to the paroxysms. Often, too, when a diminution in quantity of urine took place, it continued until several days after pain had ceased, with no very decided increase in amount subsequently, to indicate cessation of a hydro-nephrosis.[1] During the last of January large doses of potassium citrate were prescribed; this salt was continued for one month. Prior to and during this time frequent examinations for uric acid and urea were made. The result of these need not be recorded here. The urea, when potash was not administered, was usually somewhat diminished, varying between 350 and 465 grains daily, and occasionally sinking to below 300 grains. The daily elimination of uric acid was usually in excess. While taking the potassium an increase in the amount of urea and a diminution in uric acid occurred, as is usual.

Piperazin was begun on March 27, 1892, and continued steadily until July, with a single interval of eighteen days. In this time he took thirty 5-gramme bottles, in daily doses varying from 12 to 30 grains. Daily quantitative examinations of the urine as to acidity, uric acid, and urea were made preceding taking the piperazin and immediately succeeding its beginning. This was done in two periods. The second is given in the subjoined table, as the doses resorted to at first were thought to be too small to materially influence the urinary secretion. Little difference, however, actually exists as to quantity or quality of urine voided at these times under piperazin.

Estimations were now no longer made. Continued daily measurements for some time of the amount of urine passed tallied closely with the foregoing. The attacks became less frequent and severe until the ensuing (last) July, when the more violent ones ceased to occur. The patient was not seen for several months until summoned December 1st for report, prior to preparing this paper. He stated then he had noticed no blood or pus in the urine since early in June, and that none of his former attacks had recurred. At intervals, varying from a month to six weeks, he feels, for two or three days, a sensation of discomfort, not amounting to actual pain, in the loin. This same discomfort he had when I saw him. A specimen of urine was then voided, and examined after standing twenty-four hours in a conical glass. It had remained clear; was of light-amber color; non-fetid; specific gravity, 1020; contained no albumin by heat or nitric acid[2] (including overlaying a

[1] Infrequently the urine increased considerably in amount during an attack. Thus, in one twenty-four hours, 85½ fluidounces were passed. On this occasion no solids were eaten, and four quarts of milk were taken.

[2] It was possible always to demonstrate albumin by these coarser tests in the previous specimens.

cold specimen of it with the latter), but responded slightly to the ring-test with citrated pieric acid. The sediment was flocculent and very small in amount. A number of fields in three slides were examined. Pus corpuscles were present in small amount. Many fields contained none. One field held eight to ten bunched. Three round cells, apparently from the kidney, were present. One large hyaline cast and a number of casts of urates were seen; urates and urie acid crystals were numerous.

CASE II.—A. L.

Date.	Daily amount of urine in fluid ounces.	Specific gravity of mixed 24-hour specimen	Degree of acidity calculated on 100 c.c. of urine	Daily elimination of urea in grains	Daily elimination of uric acid in grains.	Daily dose of piperazin.	Remarks.
April 15	28	.....	...	....	...	None taken for 8 days.	Just recovered from an attack; urine dark red; contains much blood, pus, and amorphous urates.
" 16	32	1027	42	436	20	None taken for 9 days.	Color high; turbid; no blood macroscopically; few red disks microscopically; pus-cells in large number.
" 17	49	1022	22	378	Not estimated.	None taken for 10 days.	
" 18	68	1021	13	518	"	None taken for 11 days.	
" 19	52	.....	...	....	"	None taken for 12 days.	
" 20	62	.....	...	....	"	None taken for 14 days.	
" 22	49	1020	44	123	26	None taken for 15 days.	Slight attack.
" 23	42	1022	48	462	25	None taken for 16 days.	
" 24	48	1020	30	433	18	None taken for 17 days.	
" 25	70	1021	18	509	14	None taken for 18 days.	
" 26	50	1016	16	432	15	25 grains taken.	
" 27	58	1021	36	493	24½	30 " "	
" 28	44	1022	40	500	18½	30 " "	
" 29	34	1022	70	382	13	30 " "	Urine contains blood; slight attack; ¾ grain morphine taken.
" 30	37	1022	38	420	15	20 " "	Microscopic examination of urine, pus- and red-cells; urine still bloody.
May 1	84	1020	20	611	22	20 " "	Attack ceased April 30th; urine light color; turbid.
" 2	76	1020	13	518	26	20 " "	
" 3	48	.....	...	....	...	30 " "	
" 4	48	.....	...	....	...	30 " "	
" 5	41	1024	48	503	18	30 " "	
" 6	43½	1027	48	579	21	30 " "	Pus corpuscles; renal epithelium, bacteria; uric acid and urates.
" 7	36	1016	52	475	19½	30 " "	Attack coming on.
" 8	23	.....	...	....	...	30 " "	Preceding this table a report of the microscopic examination of this specimen is given.

The third case is in all likelihood one of mulberry calculus.

Miss A. A., first seen August 16, 1891. For four years there had been present stationary pain in the right lumbar region, occasionally accompanied

by slight paroxysms, in which the pain darted in the direction of the ureter. The pain was present intermittently at first, but had been constant in the past year. About the time this trouble began she had two quite characteristic attacks of renal colic, lasting each a day, and accompanied by bloody urine. She does not know if she then, or since, passed a stone or gravel. On examination, there was noted an extended area of renal dulness on the right, with decided tenderness on pressure over it. The left loin was normal. There was a systolic apical heart-murmur, due to mitral incompetency, probably the result of a past attack of endocarditis during rheumatic fever, which had occurred two years before. The urine was clear, specific gravity 1023, and contained a trace of albumin by overlaying it with nitric acid.[1] It was loaded with calcium oxalate and microscopic calculi of the same. There were a few pus corpuscles, red blood-disks, epithelium from the kidney and bladder. Several other specimens were examined microscopically within a short time. Calcium oxalate crystals were always present in large amount, with a few red- and pus-corpuscles.[2]

I concluded the calculus to be of calcium oxalate formation, and therefore scarcely susceptible of solution. I, however, thought it wise to make a thorough trial of a vegetable salt of potassium, as there was, of course, a possibility of the stone being composed of uric acid or urates. Potassium citrate was administered in very full doses for six weeks, totally without result. At the expiration of that time, absolutely no improvement resulting, I had Professor Keen see her, with a view to operation. Professor Keen agreed as to the diagnosis, and advised an early exploratory operation. But to this her consent could not be obtained.

Piperazin was begun in March, 1892. At first it was taken in doses of nine grains daily, and later in much larger quantities. It was continued until July. Prior to beginning piperazin the amount of urine passed was always subnormal. The specific gravity was usually high (1030 or over). The daily amount of urea was about 400 grains. The accompanying table shows that the quantity passed was uninfluenced, which continued small as before. The diminished amount constantly passed may in a measure be accounted for by the fact that the patient habitually partook most sparingly of fluids.

A number of urine examinations were made before taking piperazin and while on the drug. A second series is given in this case, as in that of the case preceding, because the dose first resorted to—nine grains daily—was thought too small to materially influence the amount or quality of the urine. The first series, however, agrees substantially with that of the second.

[1] Albumin was examined for a number of times. It was inconstantly present to Heller's test, but always responded to picric acid.

[2] A number of subsequent examinations were made. They all agreed substantially with the above, save that hyaline casts were found several times, and once granular casts, and that, after taking piperazin a week or ten days, calcium oxalate crystals were present in very much less amount.

CASE III.—MISS A. A.

Date.	Daily amount of urine in fluid-ounces.	Specific gravity of mixed 24 hour specimen.	Degree of acidity calculated on 100 c.c. of urine.	Daily elimination of urea in grains.	Daily elimination of uric acid in grains.	Daily dose of piperazin
April 9	30	1033	50	400	$15\frac{2}{5}$	6 days after discontinuance of a daily dose of 9 grains.
" 10	28	1033	46	446	$15\frac{2}{5}$	No piperazin.
" 11	32	1031	43	349	$19\frac{1}{2}$	" "
" 12	27	1030	42	321	$14\frac{1}{3}$	24 grains taken.
" 13	30	1033	14	368	20	24 " "
" 14	32	1026	34	381	15	24 " "
" 15	40	1023	28	346	15	24 " "
" 16	48	1025	33	414	8	20 " "
" 17	34	1032	54	454	15	22 " "
" 18	27	1027	38	212	$7\frac{1}{2}$	22 " "
" 19	32	1032	31	398	18	22 " "
" 20	32	1031	46	180	15	22 " "
" 21	36	1025	40	108	15	22 " "
May 1	30	1030	52	340	14	20 grains daily up to May 24, then 30 grains daily to date.
" 5	30	1030	52	400	15	30 grains.
" 6	32	1032	47	350	$13\frac{1}{2}$	30 "
" 7	37	1022	42	385	$12\frac{3}{4}$	30 "

Piperazin in this case, as regards effects on the symptoms, loin pain, etc., was most disappointing. No substantial benefit can be said to have resulted from its trial. At first it was thought improvement was occurring. The loin pain diminished and less tenderness existed to pressure. This amelioration, however, was not maintained; and now, as regards the kidney derangement, she is apparently in the condition she was prior to instituting the piperazin treatment. The loin is as tender, the aching as severe, and occasionally darting pains occur from the kidney into the hypogastrium.[1]

In the first and second of these cases practically a cure may be said to have been obtained. Undoubtedly a cure has resulted in Case I., as all symptoms referable to the kidney have been absent several months, notwithstanding piperazin was discontinued six months ago. A most interesting fact in this case is the permanent disappearance of gravel from the urine. Gravel had been a persistent and troublesome symptom for months prior to treatment with piperazin. In the second case,

[1] These were occasionally present when she first presented herself for treatment. They became a trifle more common after piperazin was begun.

one of undoubted pyelitis, in all probability calculus (uric acid or calcium oxalate), the pus has almost entirely disappeared from the urine. It now manifests its presence only by microscopic examination. None of the former attacks of lumbar pain, of seven years' continuance, have been present for seven months.

In the third case—that of probable mulberry calculus—no benefit has been obtained by the use of piperazin. This is in accordance with what might be anticipated from the behavior of piperazin to this salt of calcium, upon which it has no solvent action.

What is the action of piperazin in cases of nephro-lithiasis in which a cure is obtained? The answer seems not far to seek —by its marked disintegrating effect upon uratic stones lodged in the kidney pelvis, which it bathes in process of elimination. In the test-tube the solvent action of even dilute (1 per cent.) solutions of piperazin upon portions of uratic calculi is decided. In a warm chamber, with an equable blood-heat temperature, these are readily softened in a few hours, and this effect is more decided if the solution is permitted to flow slowly over the stone. But if calculi are so solved in the kidney, an increase in the uric acid eliminated is to be expected during their disintegration—if a stone of large size is in question.[1] This increase, though carefully examined for, was not detected in my cases. It may be that it occurred in Case I., as estimations could not be undertaken for several weeks after piperazin was begun. Then great improvement in symptoms had occurred. In the second case the doses first used were too minute for this result to be anticipated. The symptoms continuing, the remedy was withdrawn, estimations were made, and larger doses administered. No increase, however, in uric acid attributable to the remedy was evident. But also no marked diminution in symptoms of stone occurred while estimations were in progress. Distinct amelioration did occur later, while the remedy was

[1] It should, however, be here stated that a uric acid calculus may be of some size—that of a pea or larger—and weigh but a grain or two. Such a stone, requiring several days to disintegrate, would not appreciably affect the amount of uric acid excreted. A very large stone would, of course, weigh much more; evidence of its prompt solution should then be apparent.

being taken in full doses, but when the case ceased to be under continuous observation. In this case the stone, perhaps, being a mixed one of calcium oxalate and uric acid, was more difficult of solution, so that disintegration occurred too gradually to produce appreciable results.

Our present knowledge of the precise mode of action of piperazin is so limited that little more than theorizing as to the cause of the results obtained can be attempted. That piperazin is beneficial in cases of gravel and stone is certain. Sufficient clinical reports are now on record as to this. Its precise mode of action is still somewhat obscure, and has not been determined by those hitherto investigating the subject, judging from the contradictory statements, all of which are based on narrow data. Ebstein and Sprague[1] found no alteration in urea or uric acid exeretion, but noted an increase in the amount of urine, which, at times, became of an alkaline reaction. Bardet[2] reported an increase in the soluble urates, while Vogt's experiments showed a diminution in the latter, with an increase in urea. Brik[3] more recently records Heubach and Kuh's having detected a slight increase in exeretion of uric acid, while he himself noted an increase in quantity of urine; but, as with Heubach and Kuh, Brik found no alteration in the reaction, it never becoming alkaline during the administration of piperazin.

Extended examinations as to the effect of piperazin on nitrogenous exeretion in the urine should be made in both healthy and uratic subjects before accurate judgment can be arrived at as to mode of action. This I had myself undertaken, but was temporarily forced to abandon it because of pressure of other work.[4] Uric acid estimations by trustworthy methods are troublesome and consume much time.

[1] Berl. klin. Wochenschr., 1891, No. 14.

[2] Bull. gén. de Thérap., March 8, 1891.

[3] Wien. med. Blätter, December 10, 1891 (see Therapeutic Gazette, February, 1892, p. 113).

[4] Thus far I have made estimations only in cases of uric acid diathesis, in cases of gravel, and in others of suspected stone in the kidney. These were not conducted to a finality. I shall report on the subject in a subsequent paper.

In cases of uric acid diathesis other than stone, from what is known of the solvent effects of piperazin on uric acid and the solubility of piperazin urate, an increase in excretion of uric acid might be expected under full doses of piperazin. Yet some observers have reported a diminution, with a corresponding increase in urea, indicating that, besides its affinity for uric acid, piperazin promotes the transformation of uric acid into urea, as do markedly the salts of potassium with the vegetable and carbonic acids. These potassium salts were long ago asserted by Basham to act by virtue of the increased alkalinity of the blood they produce, promoting its oxidation function; and this explanation is now generally accepted. When these salts of potassium are administered, the acidity of the urine is promptly diminished, and soon disappears, while the quantity of uric acid is reduced to a minimum, and that of urea increased often several fold. Piperazin has been asserted to produce identical results on the reaction of the urine, and to also markedly augment urea elimination. I am, however, unaware of any authentic instance of the urine becoming alkaline after the use of piperazin. Perhaps larger doses than those usually administered would induce this; one-half drachm daily for some days in several of my cases failed to effect this. It may be seen by the foregoing tables that the acidity quantitatively estimated from mixed twenty-four-hour specimens was not appreciably affected by the drug. Nor can it be said that an increase in urea excretion occurred in my three cases, judging from the estimations made immediately before and after giving piperazin. In Case I., during the first five days on piperazin, subsequent to its temporary discontinuance, in which 135 grains were taken, there were only 1989 grains of urea excreted against 1603 in the five days preceding, a total increase of but 395 grains. A total increase of 14 fluidounces of urine also occurred. In Case II., 2259 grains of urea were eliminated in the first five days, and 2406 in the second, in which 135 grains of piperazin were taken, a total increase here of but 147 grains. A total diminution in urine occurred in the second five days of 2 fluidounces. In Case III., 1195

grains of urea were passed in the first three days, and 1070 in the second three days (when on piperazin in doses of 24 grains daily), a total diminution in urea of 125 grains. A total diminution in urine also occurred in the second three days of 2 fluidounces. These differences in urea excretion are, of course, too slight and common to be attributed to the administration of piperazin.[1] The extraordinary increase in urine in Case I., under piperazin, and the subsequent maintenance of the normal average, is explicable on the probable supposition I have already advanced, that of the removal of a hydro-nephrosis by rendering patent a ureter occluded by stone. In the other two cases no increase of urine occurred, which somewhat surprised me, as in several other instances in which I administered piperazin a more or less marked increase is stated to have occurred under it, though no measurements were made.

[1] In two similar periods of five consecutive days, when on no drug, a difference in the amount of urea excreted in my own urine amounted to 250 grains.

NEURITIS AND MYELITIS, AND THE FORMS OF PARALYSIS AND PSEUDO-PARALYSIS FOLLOWING LABOR.

By CHARLES K. MILLS, M.D.,

PROFESSOR OF DISEASES OF THE MIND AND NERVOUS SYSTEM IN THE PHILADELPHIA POLYCLINIC; CLINICAL PROFESSOR OF NERVOUS DISEASES IN THE WOMAN'S MEDICAL COLLEGE OF PENNSYLVANIA; LECTURER ON MENTAL AND NERVOUS DISEASES IN THE UNIVERSITY OF PENNSYLVANIA; AND NEUROLOGIST TO THE PHILADELPHIA HOSPITAL.

[Read February 1, 1893.]

Neural and spinal affections of puerperal origin, although not entirely neglected, have received but little attention in the text-books and journals. Imbert-Gourbeyre[1] refers briefly to the occurrence of paralyses from trauma during labor in a monograph on *Puerperal Paralyses*, giving several cases. One of these, from Rademacher, was an incomplete and painful paraplegia coming on at the end of a long and difficult labor, and cured in eight days, chiefly by friction. Another patient, thirty-six years old, during her third labor was paralyzed in both extremities, and recovered in a few months. Salvat is cited as reporting the case of a woman treated by him for vesico-vaginal fissure, with paraplegia produced by the long stay of the head of the fœtus in the inferior strait. Another patient, thirty-two years old, during her fourth labor, which was prolonged and the delivery by forceps, suffered great pain in the loins, accompanied by feebleness and swelling of the legs. The feebleness increased to paraplegia, and she had lancinating pains, paræsthesia, and cramps in the limbs. I have cited these cases in a paper on "Lesions of the Sacral and Lumbar

[1] Imbert-Gourbeyre: Mém. de l'Acad. Imp. de Méd., tome xxv., 1861.

Plexuses,"[1] in which I have also reported two cases of sacral neuritis, kindly furnished me by Dr. Howard A. Kelly, of the Johns Hopkins Hospital. The first patient had passed through a difficult instrumental confinement some twelve years before coming under observation. She visited many prominent gynecologists, and underwent a number of operations, the last being the removal of two large tubes and ovaries. She was relieved of menstrual exacerbations, but still suffered great pain in the pelvis, for which she was receiving galvanism, massage, and anti-lithic remedies. A careful examination showed that the uterus and its surroundings were perfectly free from disease. On making a careful rectal examination, however, outlining the sacro sciatic ligament and pyriformis muscles, and carefully palpating the roots of the great sciatic nerve, upon touching one cord she gave a sudden scream, at the same time doubling up her leg and jerking her body in the bed. Here, directly over the roots of the left sciatic nerve—the left sacral plexus—was the only diseased area which could be detected in the pelvis. All subsequent treatment was directed to this condition. The second patient had constant pelvic pain, which she described as soreness, located at the "back of the womb." She had been for several years, since the birth of her last child, under the care of gynecologists, who had not been able to give her any relief whatever. It was found by exploration that the only point of tenderness in the pelvis was at the roots of the sciatic nerve, and here she at once located all her pain, when the doctor introduced his finger into the rectum and made pressure on the nerve trunks. The case was one of neuritis. As Dr. Kelly remarks in the communication sending notes of these cases, they teach the value of exploration of the pelvis outside of the uterus and its annexes.

Ramsbotham[2] speaks of paralysis of both legs of varying degree occasionally happening after labor; more frequently when the process has been tedious and painful, but sometimes when it has been of ordinary duration, or even of unusual rapidity. He is evidently referring to the same class of cases of which we are treat-

[1] Mills: Med. News, June 15, 1889.

[2] Ramsbotham: The Principles and Practice of Obstetric Medicine and Surgery, Am. ed., 1865.

ing in the present paper. "It is not attended with cerebral affection," he says, "but is dependent on pressure which the muscles and nerves have sustained during the passage of the child's head through the pelvis. There is pain and numbness, both within the cavity and around the hip, and an inability to move the limb with freedom. It generally disappears by degrees within a few days; at other times it continues beyond the period the patient commonly remains in bed, and compels her when she rises from it to use a stick or a crutch. Fomentations in the first instance, and afterward stimulating embrocations, a *douche* or shower bath, tonic medicines, and gentle movement of the limb, will offer us the best chances of success. Hemiplegia, indeed, may appear after delivery as well as at other times; but there will then be particular symptoms, independent of those connected with the local affection, which are too well known to require mention from us here."

Cazeaux and Tarnier[1] in the American edition of their treatise, translated by Hess, under Lesions of Innervation, direct attention to the various forms of paraplegia occurring during pregnancy or labor, most of which they attribute to reflex causes. Lloyd[2] considers some aspects of the subject in a contribution to Hirst's *American System of Obstetrics.*

Winckel[3] introduces a brief chapter on the "Neuralgias and Paralyses of the Lower Limbs," with a paragraph which is comprehensive in its presentation of the etiology of these affections: "The puerperal neuroses of the lower limbs are located chiefly in the nerve trunks, more rarely in the nerve centres, and generally owe their development to parturition. Injurious pressure is effected by a large, hard, child's head, unfavorable in presentation in a small pelvis. Vigorous compression may result in complete interruption of conduction at the compressed spot. This occurs in instrumental aid during labor, inasmuch as the rim of the blades of the forceps may produce severe contusion of the sacral plexus on forced closure

[1] Cazeaux and Tarnier: Theory and Practice of Obstetrics. 8th Am. ed., edited and revised by R. J. Hess, M.D., 1887.

[2] Lloyd: Hirst's System of Obstetrics by American Authors, vol. ii.

[3] Winckel: A Text-book of Obstetrics. Translated by J. Clifton Edgar, A.M., M.D., 1890.

as well as during extraction. The thick nerves are also compressed not unfrequently by a pelvic exudation, or small extravasations from the neighboring parts extend to the sheathes of the nerves, or hyperæmia or œdema of the neurilemma appear spontaneously. We have already mentioned that parametritis may give rise to such neuralgias. The location of the affection is in the external and middle cutaneous, obturator, or sciatic nerves. The two latter nerves are especially apt to suffer during labor. Even the slight pelvic exudations, for example, one resulting from phlebitis, may make a nerve trunk incapable of conduction without compressing the entire trunk. (Leyden.) Injuries of the vagina, with subsequent severe cicatricial contraction, may also exercise traction and pressure on individual nerve trunks of the pelvis minor, and hyperæsthesia and motor disturbances may thus be produced."

Brief references to this subject are also to be found in some of the neurological text-books, but it is not in these treated of systematically.

To Dr. Anna M. Fullerton, of the Woman's Hospital of Philadelphia, who, in her large obstetrical experience, has seen a number of instances of paralysis and pseudo-paralysis during the puerperium, I am indebted for the notes of some cases which will be given later. In a letter accompanying these notes she says that partial paralysis is in general short-lived, being limited generally to two or three days. She has had paralysis of the rectum and sphincter from long-continued pressure of the head upon the floor of the pelvis, which sometimes lasted for a week or ten days, and which was only overcome by faradism and time; also vesical paralysis, which has sometimes lasted up to the sixth, seventh, or twelfth day after delivery. According to Dr. Fullerton, cases of contracted pelvis, which, because of a narrow inlet, have resisted the engagement or descent of the head, and cases in which for any cause there has been impaction of the fœtal head, have been those most commonly resulting in paralysis.

The following are her notes of one of these somewhat serious but comparatively short-lived cases. Probably the case was one of pressure neuritis.

M. M., married, a dwarf, aged twenty-five years; pubis generally contracted. The patient came to the Woman's Hospital maternity, September, 1892, for her second confinement. The first stage of labor was very slow; descent of the fœtal head very slow, with impaction in the pelvis before rotation; presentation, vertex, second posterior variety. There was threatened asphyxia of the child with exhaustion of the mother, which led to forceps delivery. The extraction was easy, and no laceration of the soft parts occurred. She complained, the night following delivery, of great pain in the right groin and entire right limb, and was unable to move the limb. Pressure on the anterior crural and obturator nerves caused her to cry out with pain. Flaxseed poultices were applied over the groin; the limb was elevated and bandaged with flannel. These symptoms lasted about ten days, and then entirely disappeared. She was out of bed by the third week, and able to return to her home the fourth week.

The most valuable recent paper on puerperal paralysis of peripheral origin is that of Hünermann,[1] assistant in the clinic of Gusserow at Berlin, on "Paralysis in the Region of the Sciatic Nerve following Labor." On examination of this paper, which I have obtained quite recently, I find that Hünermann has reasoned with reference to the production of the most common type of these palsies in much the same manner as I had already presented the subject in the arena at the Philadelphia Hospital and to my ward classes. He carefully traverses the literature of the subject, and his paper can be consulted with advantage for French and German references. Among the writers to whom he refers are Von Renz, Koeppen, Von Leyden, Kast, Möbius, Von Basedow, Gerber, Breisky, Litzmann, Kehrer, Dorion, Bianchi, Lefebre, Brivois, Guinon and Parmentier, and Handford, to some of whom I shall have occasion to allude.

The affections to which attention will be especially directed in the present paper can be conveniently discussed under five heads:

1. Traumatic paralysis of the peroneal type, usually associated with severe neuritis.

2. Sacral and sacro-distal neuritis, sometimes accompanied by a pseudo-paralysis, and often maintained or aggravated by disease and displacements of the pelvic organs and tissues.

[1] Hünermann: Archiv für Gynäkologie, Berlin, 1892, vol. xlii., part 3.

3. Puerperal neuritis, local or multiple, and due to septic or other infection.

4. The neuritis, paralyses, and pseudo-paralyses of phlebitis, and phlegmasia alba dolens, which are often septic, but have special features.

5. Puerperal myelitis occurring under the same conditions as the forms of septic and infectious neuritis.

Brief reference will also be made to forms of hysterical and reflex paralysis which must be diagnosticated from the affection under consideration. Some cases illustrative of each of the classes mentioned will be presented, the first class, however, receiving the fullest discussion. The first three cases are interesting examples of this type of paralysis and neuritis.

PARALYSIS OF THE PERONEAL TYPE AND ASSOCIATED NEURITIS AFTER LABOR.

Case I. *Prolonged labor and forceps delivery; left peroneal paralysis, anæsthesia, and neuritis; recovery from the neuritis and persistence of the paralysis; previous history of syphilis.*—The notes of this case were presented to the Philadelphia Neurological Society, and, with the discussion, were published in the *Journal of Nervous and Mental Disease*, February, 1892. I will give them here somewhat condensed.

Mrs. W., aged thirty years, had been twice married, and believed that she had been infected with syphilis by her first husband in 1884. In 1889 she married again, and was in labor June 3, 1891. The labor was prolonged and painful; instruments and chloroform were used. On recovering from the anæsthetic she felt a severe pain in the left hip, leg, and foot. The limb was paralyzed, and from this time she suffered with all the symptoms of a severe neuritis, which continued with little abatement for about three months, when it began to decrease. In October it had almost entirely disappeared, but the left leg was still in an extreme condition of motor paralysis.

Five weeks before she was examined by me she was suddenly stricken, without unconsciousness, with an attack of paresis in the left upper extremity, and some difficulty of speech, from which, however, she recovered. The attack was cerebral, and has no bearing upon the problems discussed in this paper, except in so far as syphilis was probably the predisposing cause of both conditions.

On examination it was found that all the movements of the muscles below the knee supplied by the branches of the external popliteal or peroneal nerve were paralyzed and non-responsive to electric currents. The

foot was swollen, purplish in color, and cold. No loss of sensation was found anywhere, but she still had great pain on pressure at the sciatic notch, and in such cords of the sacral plexus as could be reached by the rectum. The muscles above the knee, and also the gastrocnemius, soleus, and posterior tibial, were not affected. Knee-jerk and muscle-jerk were normal on both sides. Bladder and rectum were not involved.

In the discussion of this case, reported in the *Journal of Nervous and Mental Disease*, February, 1892, Dr. Wharton Sinkler spoke of a case which had fallen under his care. The patient was a small woman, a primipara, and the labor was instrumental and very difficult. On recovering from the anæsthetic she complained of severe pains in the legs, and the pain and hyperæsthesia continued for several weeks. He saw the patient in consultation about six weeks after labor. There was then contraction of the knees, and the muscles were considerbly atrophied. Extreme hyperæsthesia was present, especially below the knee, but no special pain over the nerve trunks, no loss of reflexes, and no loss of response to the faradic current. The patient recovered entirely, and in two months was walking perfectly well. The neuritis was complete and equal in both legs.

In the same discussion, Dr. F. X. Dercum reported a case which he saw some months after labor. Great violence had been used. Paralysis and wasting were both present. The type of the paralysis was not specified in either of these cases.

Case II. *Left peroneal paralysis and anæsthesia; neuritis after two weeks; recovery from the neuritis; persistence of some paralysis, but very great improvement.*—Mrs. H., aged thirty-three years, was confined May 23, 1892. She was in labor twenty-one hours, with severe, almost constant pain during the last ten hours. She was under the influence of chloroform for nearly an hour, and was delivered by forceps. After delivery she found that her left leg was paralyzed and anæsthetic, requiring to be lifted for all purposes. During the third week pain began in the hip, and rapidly became more and more severe, extending down the legs to the toes. Soon the other leg was attacked with a similar but less severe pain. Sometimes the pain was darting and burning, and the left foot was extremely sensitive to handling. Some loss of power was present in the right leg. She continued to have great pain, and was only able to stand or walk with the aid of a cane, and with much suffering on making the efforts. Her treatment consisted of partial rest, the use of anodynes with liniments externally, and the internal use of morphine, atropine, and occasionally the bromides. Hypodermatic injections of morphine and atropine were necessary for some time; but the morphine had unpleasant constitutional effects upon her, causing nausea and dizziness, so that it could not be continued.

This patient was first examined by me early in September, 1892. Her left leg was extremely sensitive over both its anterior and posterior aspects, especially above the knee in the left popliteal space. On the right side similar

hyperæsthesia was present, but not nearly so marked. Lateral squeezing of the left foot caused considerable pain, which was absent in the right. Examination both by the rectum and vagina showed extreme sensitiveness of the nerves of the pelvis, particularly on the left side. Until a week or two before coming under observation, moving about and sitting down with a slight jar would cause pain, which, she said, "seemed to be in the bones on each side of the rectum." No paralysis was present in the right lower extremity. The movements of this leg were almost perfect, showing only a little weakness. Below the left knee she was totally paralyzed for the movements of dorsal flexion at the tarsus, and of extension of the phalanges of the great toe and of the other toes. Movements controlled by the posterior muscles of the leg below the knee were present.

Believing that she had suffered from a crushing of the lumbo-sacral cord and upper sacral nerves and a consequent neuritis, and that this neuritis was still present and active, although improved, she was placed in bed with sandbags to her limbs; and upon the use of hot douches, mercurial and atropine inunctions, salicin and other remedies internally, in accordance with a plan of treatment which will be discussed later. After the neuritis had largely subsided, galvanism, massage, and Swedish movements were systematically employed. The patient returned to her home cured of her neuritis, and with only partial paralysis of her anterior tibial remaining. She is still treated at her home with galvanism.

Case III. *Probable neuritis before labor; prolonged labor and forceps delivery; neuritis and right peroneal paralysis; recovery from neuritis, but persistence of some paralysis.*—Mrs. R., aged twenty-four years, had been healthy before her marriage. For a week before her first child was born she suffered severe pelvic pain. Labor was prolonged, and she was delivered with instruments. Before delivery she complained of severe pain commencing at the hip and extending to the toes of the right foot; and just at the time of the birth of the child she felt a very sharp pain commencing at the hip and extending downward to the toes of the right foot. After delivery the pain and tenderness spread through the entire limb, every movement and jar of the leg causing suffering. On the ninth day she attempted to get on her feet, but found that the right leg was nearly helpless, and the pain on movement was so great that she was forced to lie down. This condition continued for five weeks. She thought the pain had been most severe on the outside of the limb from the ankle to the hip. At the end of five weeks she was able to walk, but found that she had not the proper use of her foot. She was not aware of having had at any time loss of sensation.

She came as an out-patient of the Polyclinic Hospital, remaining for a few weeks, and then disappearing from the service. She was sent for, however, and examined by me again January 28, 1893. In the meantime she had had another child, October 15, 1892. The condition of her leg was practically the same as at her first appearance at the Polyclinic service, although the

woman herself claims that she had had better use of her limb since her last confinement. While having the second child she experienced pain similar to that from which she suffered during the birth of the first child, but of less severity.

She has at present no sensory symptoms. The right leg below the knee is a little smaller by measurement than the left; circumference of the left calf is 13.5; of the right, 12 inches. The movements now completely paralyzed are those of the tibialis anticus and peroneus tertius muscles. Dorsal flexion of the great toe and of the other toes can be performed, but flexion at the tarsus cannot. The extensor longus digitorum and extensor proprius pollicis muscles are therefore at present not paralyzed. Faradic and galvanic contractility are abolished in the anterior tibial and peroneus tertius—in fact, these muscles have largely disappeared.

A summary of Hünermann's four cases will be of interest in connection with these reports:

His first case was a primipara, aged thirty-six years. Labor began January 22, 1892, in the morning. On the 23d, she had severe pain and convulsive movements of the right leg. Forceps delivery under an anæsthetic was unsuccessfully attempted, when she was transferred to Berlin, a few miles distant, arriving January 25th, at the Charity Hospital, after three and a half days' labor. Details of her condition are given by Hünermann. Perforation and extraction were performed. The child had evidently been dead for some time, but the puerperium passed favorably. On the morning after confinement, Hünermann discovered complete paralysis of the right peroneal nerve. The woman complained of severe pains in the course of the sciatic, also of a feeling of numbness on the outer side of the left calf, and of tingling and creeping sensations in the toes. She could not dorsalflex the right foot or extend the toes, nor sink the inner border of the foot: but she could with considerable force bend the foot downward; she could also flex the toes and execute all the movements of the knee- and hip-joint without difficulty. There was not the slightest disturbance of sensation. During the first three nights her sleep was completely interrupted by the pains in her legs. Wrapping the leg in cotton and bandaging it gave some relief. From the fourth day the patient was treated with the faradic current; but when on the thirteenth day she got up, the muscles supplied by the peroneal were completely paralyzed. On the sixteenth day she was carefully examined, and depression of the electrical excitability of the paralyzed muscles was established. She received a daily bath and electricity. The impairment in motility was still noticeable at the end of the third week, when the patient left the hospital. Her walk was still greatly affected.

Hünermann's second case was a primipara, aged sixteen years, of frail bony development and childish in looks. Measurements were taken, which

showed a contracted pelvis. The patient was at the end of her first normal pregnancy. The child was in the first position on admission, January 22d. Details are given of the labor, which was tedious and halting, the pains becoming stronger until the 25th. The head had entered the pelvis with a remarkably low position of the fontanelle. Toward evening the patient felt pain and a feeling of numbness and tingling in the right calf downward from the knee; and she could not step on the right foot when she got up to pass water. She was delivered January 25th, under an anæsthetic, with the forceps, of a slightly asphyxiated girl, well developed, and showing a long line of pressure, which began about the right frontal eminence at the border of the hair, and reached diagonally to the left and backward. Hünermann particularly mentions this pressure-line, which only disappeared on the tenth day, and was in his opinion not caused by the forceps, but by the linea innominata, which had resisted the progress of the child's head. On the first day of the puerperium the mother was found to have a complete paralysis of the peroneal nerve. As in the previous case, the phenomena were the same during the first few days, being without disturbance of sensibility, but with complete loss of function of the groups of muscles supplied by the peroneal nerve. She complained of painful twitching and of numbness in the right calf. On the evening of the first day fever set in, lasting until the thirteenth day. The perineum was ruptured; involution of the uterus was faulty, and the lochia of bad odor until corrected by the antiseptic douche. On the seventh day and later she developed great pain in the right trochanter, and other symptoms of a descending neuritis. She suffered from headache and insomnia. She left her bed on the fifteenth day. On the twentieth day, examination showed unequal pupils, not reacting to light, and not contracting on convergence; knee-jerk was missing on both sides. The ocular and knee phenomena are referred by the reporter to an attack of diphtheria which the girl had had in her ninth year; or, perhaps, to congenital syphilis. She undoubtedly, however, had paralysis of the right peroneal nerve, which only developed in connection with or following the labor, although Hünermann says that it must be admitted that a previous disturbance of her central nervous system had predisposed her to an affection of the peripheral nerves. Several weeks after labor, only a slight improvement in the peroneal paralysis had been accomplished.

The third case of Hünermann was a well-built woman with normal pelvis, aged thirty-four years, who had given birth to five living children, without artificial aid. She was delivered in twelve hours, under chloroform, with forceps. The child was dead. Immediately after reaction from the anæsthetic she felt strong cramp-like pains in the left leg. She continued constantly to complain of pain in this leg, but Hünermann discovered a complete paralysis of the muscles supplied by the peroneal nerve of the left leg, while tactile sensibility was undisturbed. She had a hæmatoma in the left wall of the vagina, which disappeared in about two weeks. No pelvic exudate could

be found. Paralysis, with reactions of degeneration in the muscles supplied by the peroneal nerve, continued. An examination made ten months after labor is given. The left leg was then somewhat atrophied on the anterior and outer side. The sciatic was not especially sensitive at the great sciatic notch and trochantor major upon pressure; but distinct pain was present when the head of the tibia was pressed. Knee-jerk was more marked on the left than on the right. Sensibility was not disturbed. This case of paralysis Hünermann believes was due to the application of forceps, and as favorable to this view was the fact that the woman at once after the operation felt the pain in the left leg. A pressure of the roots of the sciatic nerve could easily take place; again, it was the lumbar roots which especially suffered while the sacral nerves were very little affected. The case reminds him of one of the cases reported by Bernhart, of left-sided peroneal paralysis after forceps delivery. In this case, also, forceps were applied in the high position, and the second position of the head.

The fourth case, M. R., aged thirty-seven years, had gone through four hard confinements, all the children being born dead; she had had three forceps deliveries. At the last confinement, after application and use of the forceps, she had a sudden tearing pain in the left side of the pelvis; at the same time she had strong jerking in the left leg. Since then she had been paralyzed in the same leg, and shortly after confinement she had had wearing and jerking pains in this leg. She had no fever or parametritis after labor. Being disabled from walking, and the treatment at her home not being satisfactory, she came, six months after her confinement, to the medical clinic of Dr. Goldscheider, where Hünermann had the opportunity of examining her. He found an otherwise healthy woman who showed no signs of disease of the internal organs, with a marked paralysis in the region of the peroneal nerve, while the muscles of the tibial nerve were also affected. The paralyzed muscles were distinctly atrophied. Her walk, though awkward and clumsy, was quite possible without apparatus. Electrical examination showed complete reaction of degeneration in the left peroneal region; and in the region of the left tibial a slight quantitative change to both currents was present.

I have thought it worth while to summarize these cases of Hünermann in connection with my own records, as the subject is one that has not been discussed in detail by writers, and, in particular, the type of paralysis which so often results has not been pointed out, so far as I know, except by Hünermann.

In the cases described or referred to by Imbert-Gourbeyre, Ramsbotham, Winckel, Lloyd, Kelly, Fullerton, and others, no reference to any special form of paralysis has been made.

This question of the particular type of paralysis which results,

is one of the most interesting connected with the subject in hand. It will be recalled that my three cases were left with persistent paralysis in all, or some of the muscles supplied by the branches of the external popliteal or peroneal nerve. The cases of Hünermann were also of this type, and his paper is largely devoted to an exposition of the anatomical reasons for the occurrence of this particular form of paralysis. I had arrived at almost identical conclusions with those of Hünermann before I saw his paper, but I am indebted to him for interesting details and particular points in the anatomical explanation, of which I shall make use.

The sciatic nerve arises from the apex of a triangle formed by the sacral plexus at the distal border of the pyriformis muscle. The plexus lies on the anterior surface of this muscle in such a manner that the first sacral lies above the proximal, and the third under its distal edge, the second sacral being between the two on the anterior surface of the muscle. While the plexus thus lies with its sacral roots on a soft layer of muscle, the lumbo-sacral nerve (lumbo-sacral cord), rising from fibres given off in part from the fourth and wholly from the fifth lumbar nerve, glides almost immediately upon a bony base over the sharp linea innominata. This lumbo-sacral nerve is mainly the root for the peroneal or external popliteal nerve. The superior gluteal nerve arises from the posterior part of this lumbo-sacral cord; and, therefore, we would expect to and do find it affected in some of these cases of puerperal paralysis.

Lefebre, among others, has proved that the peroneal or external popliteal nerve is a continuation of the lumbo-sacral cord; and rare cases have been reported in which it could be traced as a separate nerve all the way from the pelvis.

A study of this kind shows how anatomico-physiological investigation and practical medicine sometimes reciprocate their services. One of the important problems in spinal and peripheral localization is the determination of the relationship of particular segments of the spinal cord to certain nerve movements, reflexes, and areas of sensation. M. Allen Starr[1] has published a table of the localization of the segments of the spinal cord, which has been modified

[1] Starr: Amer. Journ. Neurol. and Psych., 1884, and Amer. Journ. Med. Sci., 1888.

and added to by me in a paper on "Spinal Localization."[1] The results embodied in these tables were arrived at by physiological, clinical, clinico-pathological, anatomical, and histological investigations. In the present study practical obstetrics assists us to a decision as to the segmental origin, root, and exact course of one of the most important nerves of the lower extremity.

Below is given from this table that portion which relates to the localization of the representation of movements of the lower limb in the spinal cord from the fourth lumbar to the second sacral segment:

Fourth Lumbar Segment:

Muscles.—Abductors of thigh.
Adductors of thigh.
Flexors of knee.
Tibialis anticus.
Peroneus longus.

Fifth Lumbar Segment:

Muscles.—Outward rotators.
Flexors of knee.
Flexors of ankle.
Peronei.
Extensors of toes.

First and Second Sacral Segments:

Muscles.—Flexors of ankle.
Extensors of ankle.
Intrinsic foot muscles.

The chief divisions of the peroneal are the anterior tibial and musculo-cutaneous nerves, the former supplying the tibialis anticus, extensor longus digitorum, peroneus tertius, and extensor proprius pollicis muscles. One of the branches of the anterior tibial also supplies the extensor brevis digitorum; and from the musculo-cutaneous nerve are given out fibres to the peroneus longus and brevis. Any or all of the subdivisions of the peroneal may be affected in

[1] Mills: Therapeutic Gazette, May 15 and June 15, 1889.

these traumatic puerperal palsies, but the movements controlled by the anterior tibial are most likely to remain persistently paralyzed, as a brief reference to the movements effected by these muscles alone, or in groups, in connection with a study of the details of the cases, will serve to make clear. The tibialis anticus muscle elevates the inner front portion of the foot, flexing it at the ankle-joint and adducting it. The extensor longus digitorum, commonly working with the extensor proprius pollicis, extends the toes and, continuing, helps to flex and abduct the foot. The peroneus tertius is really a part of the extensor longus digitorum, and works with the anterior tibial in the direct flexion of the tarsus upon the leg. The peroneus longus and peroneus brevis, which are supplied by the musculo-cutaneous, evert and rotate the foot outward.

A study of these facts of the table shows that the movements paralyzed in these cases of puerperal traumatism are those of the muscles supplied by the peroneal or external popliteal nerve, and that these movements have their representation chiefly in the fourth and fifth lumbar, and the first sacral segments of the spinal cord, where the lumbo-sacral cord and first sacral nerve evidently arise.

It is a fair question whether the spinal cord does not become secondarily diseased by extension of the inflammation backward along the great nerve cords assaulted. The paralytic, trophic, and vasomotor phenomena presented by some of the cases are distinctly those of localized poliomyelitis, but these are difficult to distinguish from the conditions due to a complete crushing of a large nerve root or cord.

While a complete crushing of the lumbo-sacral cord and of the upper sacral nerves would account for a long and persistent palsy of the muscles supplied by these nerves, it is not unlikely that the neuritis in some of these cases has not only extended and diffused itself throughout the extremity, but has ascended and entered the cord, and attacked the ganglion cells of the cornua, giving us in a word, a neuro-myelitis.

In connection with this point the method of the extension of the inflammation from one limb to the other is of interest. This occurs after a longer or a shorter time; the two limbs are not coincidently affected with the inflammation. It is probable that the inflamma-

tion extends not in the pelvis nor by way of the cord proper, but rather by the cauda equina, spreading sidewise from one closely apposed nerve, or nerve-sheath, to another.

Although these peroneal palsies usually occur in cases of instrumental delivery, the traumatism which produces them is not commonly inflicted by the forceps. The labor is instrumental because, owing to a contracted pelvis, it is greatly prolonged. The injury to the nerve cords is usually inflicted by the skull of the child, although nerve injuries are doubtless sometimes inflicted by instruments. Hünermann speaks of this being the case sometimes when pendulum, side to side, and rotating movements are made. One practical point in the diagnosis of nerve injuries made by the forceps from those caused by the head of the child is that in the former the second and third, and even lower sacral nerves, are most likely to be injured, and in the latter, the lumbo-sacral cord and first sacral nerve. Cases have been reported in which, from instruments, the posterior calf muscles—those supplied by the internal popliteal—have been paralyzed instead of those supplied by the external popliteal or peroneal. In one case which has come to my knowledge, a sharp pain came on immediately after the application of the forceps, and the patient said that this pain was much greater than any endured during the prolonged labor. She was at the time partially under the influence of ether, but cried aloud with the pain in her leg when the forceps were applied. The pain, she said, was different from the ordinary labor pain, although in her case this was extreme. Afterward she had some neuritis, which continued for six weeks, and when she was up and walking about she was quite lame for at least two weeks. This was probably an instance in which some portion of a nerve had been pinched—one of the cords of the plexus, and probably the second or third.

From several points of view prophylaxis is of great importance in these traumatic cases. Certain obstetrical questions need to be more fully considered than can be done by one who, like myself, is not an obstetrician. The reports of the cases by Hünermann have a value not possessed by the records of my own cases, in that he gives certain important obstetrical details, such as the positions of the head and pelvic measurements. These measurements, accord-

ing to him, in some of the cases showed a generally contracted pelvis. Certainly these cases emphasize the importance of taking pelvic measurements. Instrumental delivery should, if possible, be effected comparatively early; or, even the propriety of resorting to perforation or Cæsarean section should sometimes be taken into consideration. The delay in using forceps, rather than the forceps, is sometimes responsible for the nerve-crushing, neuritis, and paralysis.

According to Hünermann, these accidents should not occur in normal pelves, except in face and brow presentations. He quotes two cases from Gerber, in one of which the presentation was of the face, the brow forward and to the right.

It is a striking fact that two sisters of one of my patients died in labor within two months of her own delivery. The three sisters may all have had generally contracted pelves. In the simple flat pelvis and the rhachitic flat pelvis, according to Hünermann, if instruments are not used, paralysis is not likely to occur, the oblique diameters of the pelvis being rather above than below the average in these cases.

SACRAL AND SACRO-DISTAL NEURITIS WITHOUT TRUE PARALYSIS, ALTHOUGH SOMETIMES ACCOMPANIED BY PARTIAL OR BY PSEUDO-PARALYSIS.

During labor, especially when prolonged, and sometimes even during the last stages of pregnancy, as the result of friction and pressure, a lumbo-sacral neuritis of varying intensity arises, and may continue for a longer or shorter time according to the constitution of the patient, the state of the uterus and other pelvic organs, and the course which is pursued in the management of the patient. Some of the cases which are alluded to by Dr. Fullerton as instances of short-lived partial paralysis are doubtless of this class, as, for instance, the one of which brief notes have been given in the introductory portion of this paper. This woman suffered great pain in the groin and limb, and was unable to move the latter, and pressure on several nerve trunks caused much suffering. In three weeks, however, she was practically well, the pain and paresis

having disappeared in ten days. Every general practitioner will have in mind some case of this character which has recovered under rest, and which rest has been compelled by the patient's suffering as much as by the advice of the doctor; or the case has recovered without rest or treatment through the robust recuperative powers of the individual; but not a few patients of this class are practically neglected, all the conditions present not being fully recognized, and the treatment, as a rule, not being sufficiently painstaking and thorough.

Altogether similar in their symptomatology are some cases, which appear to have no immediate connection with the processes of pregnancy or labor, but are due to the irritation which is maintained by the nagging of an enlarged or displaced uterus, or by disease and enlargement of the ovaries and tubes, or by inflammatory processes in the pelvis, no matter how they may have originated. I wish, however, especially to direct your attention to a set of cases which seem to date back to a particular labor, although they have evidently been aggravated and perhaps continued by conditions which may or may not be associated with the puerperium. I could give notes of many minor, although sufficiently troublesome cases of this description, but I shall content myself with presenting the details of one case in which the symptoms were severe and striking. This history will be presented without special comments, as the details speak clearly for themselves.

Case IV. *Sacral neuritis; pseudo-paralysis; anæmia and neurasthenia.*—W., aged thirty-three years, had never been very strong, and when a child had been subject to precordial and other neuralgic attacks. Her mother had been subject to headaches and neuralgic attacks, and her only sister had had chorea as a child. Menstruation started when she was twelve years old, and had always been free, lasting usually from five to seven days. She was almost continuously pale and anæmic-looking. She had had three children. Six years before coming under observation, while carrying her first child, she began to suffer with pain from the heel to the hip of the left leg; this never entirely left her, and with each child she had become worse. After the birth of the third child, about one year before she was first seen, it became more severe, and she was compelled to use crutches for a month, chiefly because of the suffering it caused her in attempting to walk. At frequent intervals also, she had exacerbations of pain in the left leg, and paroxysms of pain in the back, usually compelling her to go to bed. Her

suffering was so great, and at times the limb was so helpless, that she could not move it.

Careful examination showed great sensitiveness, particularly over the sciatic distribution, although not confined to it. The right lower extremity was also sensitive to pressure and handling, but not nearly to so marked a degree. Examinations were made both by the rectum and the vagina, and revealed extreme sensitiveness over the left sciatic plexus, less marked on the right side. The uterus was found to be prolapsed, enlarged, and completely retroverted, with pelvic exudations and adhesions. The os was patulous, and slight cervical and perineal lacerations were present. The ovary and tube on the left side were tender and somewhat enlarged; the same conditions, but much less in degree, were present on the right. The uterus and surrounding tissues were all extremely sensitive. In brief, the nerves and the other organs and tissues in the pelvic cavity were in an inflammatory state, much more decided on the left. Treatment was instituted with the threefold purpose of improving the general health, of relieving the neuritis, and of restoring the uterus and its appendages as nearly as possible to a healthy state. She was placed in bed, with sand bags to her limbs; mercurial inunctions with atropine were used twice daily; strychnine, salicin, and iron were given internally, with at times other tonics and nutrients and antineuritic remedies; hot douches were used to the limb twice daily, and she was placed upon a nourishing and digestible diet. Phenacetin and sulphonal were occasionally used for pain and sleeplessness.

The gynecological treatment was carried out by Dr. M. I. Bassette. Douches of hot water with opium and glycerin were used nightly; every other day for a time vaginal tampons, medicated with belladonna and boro-glyceride, and later with a ten per cent. solution of ichthyol, were introduced. Gradually the exudate diminished, and the uterus became smaller and movable, when it was replaced daily, and the patient was made to assume the knee-chest position twice a day. The sensitiveness, both internal and external, steadily improved, and the attacks of paroxysmal pain became less and less frequent.

After six weeks of treatment a consultation was held with Dr. B. F. Baer. The uterus was still enlarged, with a tendency to retroversion, unless held in position with tampons. It was decided to curette with a view to decrease its size and lessen the tendency to engorgement. After the operation the uterus was thoroughly washed out and injected with carbolic acid at intervals of a few days. She was also subsequently treated for three weeks with carbolic acid and iodine, at intervals of a few days. She was also given ergot and nux vomica, and under this treatment the uterus became smaller and less vascular, and remained in position longer without support.

After three months the patient returned home, the neuritis having disappeared, and the uterus and its appendages in such condition as to require only occasional treatment.

In all cases of this kind, and even in cases in which the symptoms are much less striking, careful examination should be made by the vagina and the rectum. The finger, guided by certain well-known landmarks, can reach some of the cords of the sacral plexus. Pressure upon or even a gentle touch of the inflamed nerves will cause excruciating pain. Any nerve pressed or rolled on a hard substance like bone, will of course be a source of pain, but the suffering which is experienced in cases of true neuritis is of scarcely endurable character. With a little practice we become more and more skilful in reaching the nerves and in distinguishing between the pain of inflammation and an ordinary hurt of the nerves.

The large nerve cords of the sacral plexus are not alone inflamed in these cases, but the inflammation may spread to the nerves distributed everywhere in the pelvis, and to the nervous plexus to the uterus, which, Rein[1] has shown, lies mainly in the cellular tissue surrounding the vagina, at the point where the hypogastric plexus anastomoses with the filaments of the sacro-uterine nerves. All the fibres which go to the uterus, either from the hypogastric plexus or from the sacral nerves pass through this plexus. This accounts for the extreme sensitiveness of such patients, even to careful vaginal examination.

One mode in which the post-puerperal paralysis may develop is through inflammatory processes originating anywhere or anyhow in the pelvis. Hünermann refers to this cause and cites a few cases. Intra-pelvic exudation may be of such size as to cause pressure, or the exudate may involve the lumbo-sacral cord or sacral nerves. To such cases I have also called attention in my former paper on "Sacral and Lumbar Lesions." Hünermann refers to three cases reported by Von Leyden, of severe sacral neuralgia, all having high fever. In one of these cases an autopsy was had and showed peritonitis, pleuritis, and numerous thrombotic veins in the pelvis, one of which ran close to the sciatic and was bound to it by infiltrations. Microscopic examination showed both peri-neuritis and neuritis. Von Dorion has described a case after labor with abscess in the left pelvis and peroneal paralysis.

[1] Rein: Société de Biologie, quoted in American Journal of Obstetrics, vol. xvi., 1888.

PUERPERAL NEURITIS DUE TO SEPTIC OR OTHER INFECTION.

In a third class of cases, puerperal neuritis, isolated, diffused, or multiple, and probably infectious in origin, is present. A few writers have reported cases of multiple neuritis shortly after normal labor. Auto-infection has been suggested as the best explanation. Some of these cases occur during pregnancy, thus indicating their non-traumatic origin. In some the neuritis and consequent paralysis has been in part or chiefly in the upper extremities, as in cases reported by Möbius and Kast. The symptoms present need not be detailed; they are in brief those of neuritis, local, diffused, or multiple; pain, hyperæsthesia, paræsthesia, paralysis, or pseudo-paralysis; sometimes anæsthesia; often changes of the reflexes; cramps and contractures, and occasional atrophies and reactions of degeneration. Usually these cases occur in the first, second, or third weeks after labor, but they may follow immediately or at a later period than three weeks. Theoretically no good reason exists why, as the result of infection either from without, or perhaps from within, any form of puerperal neuritis may not occur, and it is of some practical importance to separate traumatic cases from those which have a septic or infectious etiology.

Möbius[1] has reported the case of a woman, aged twenty-nine years, who, two days after arising from bed, and three weeks after the birth of her child, had a sense of painful cramp in the left calf, which kept her in bed for three weeks, at the end of which time some enfeeblement of movement of the forearms manifested itself. A few days later there was complaint of pain in the scapular regions which lasted for a week. Then, in the ninth week, peculiar sensations in the right forearm were perceived; and the act of writing could not be performed, because the thumb failed to firmly grasp the pen. The flexor longus pollicis was wasted and presented reactions of degeneration. Electrical treatment was followed by little improvement. In a second case a woman, fifty-five years old, complained of pain at the right elbow, which readily yielded to ordinary measures. Examination, however, disclosed the fact that atrophy of the ulnar and thenar muscles had been present for thirteen years, first showing itself shortly after labor. Motility was not materially impaired, and electric reactions were presented. Both

[1] Möbius: Münchener medicin. Wochenschr., 1892, No. 45, p. 799.

cases were considered to be instances of neuritis of puerperal origin. Möbius[1] also records the case of a patient who had had a perfectly normal labor and the puerperium free from fever. She first complained of pain, and paræsthesia in the upper extremities, with diminution of motor power; two weeks later the same symptoms were present in the legs; no anæsthesia; reflexes normal, as were also the responses of the muscles to mechanical irritation; the muscles were somewhat atrophied in the upper extremities, but not in the lower. Recovery occurred under treatment by iodide of potassium. Later she had a mild form of neurasthenia, but without any of the above symptoms of neuritis, except tenderness on pressure over the brachial plexus. Möbius considers the initial localization of the illness as characteristic, for in the later course all forms of neuritis may become general and give us the same picture.

Handford,[2] in a brief communication on the puerperium as a factor in the origin of multiple neuritis, has reported three interesting cases. Two of these bear out his diagnosis of multiple neuritis. In both, alcohol was a predisposing factor. As the notes of these cases are brief I will give them.

K. M., aged forty-three years, married seven years, three children, has always been a stout, florid, healthy woman. Her husband kept a public house, but evidence of drinking habits in the patient could not be obtained. Three days after her confinement she lost power in the legs, and at the same time felt strange sensations like pins and needles in the arms. This was soon followed by paralysis. She had been able to walk about up to the date of her confinement. The general course and character of the paralysis was typical of an alcoholic multiple neuritis. There was loss of muscular sense, impaired cutaneous sensibility, but greatly increased deep sensibility, great muscular wasting, loss of knee-jerks, and the presence of the reactions of degeneration in the muscles of the legs and arms. The condition of the eyes was normal. She was able to walk a little in six months, and in twelve months was comparatively well.

The second case was a stout, well-nourished woman, aged thirty-four years, also the wife of a public-house keeper. She suffered with complete paralysis of the extensors of the toes and the flexors of the ankle, with weakness of most of the other muscles of the leg, coming on immediately after confinement. The ankle-joints were habitually extended and the toes pointed. There was much hyperæsthesia, both superficial and deep, and a little œdematous swelling. No contraction could be elicited in the muscles below

[1] Referred to by Hünermann. Abstracted in Journal of Nervous and Mental Disease, January, 1891, vol. xvi., No. 1, p. 45.

[2] Handford: Brit. Med. Journ., November 28, 1891, vol. ii. p. 1144.

the knee by as strong a faradic current as the patient could bear. A voltaic current from twenty-six cells, registering thirty milliampères, caused much pain and some contraction of the rectus femoris, deficient in quantity and normal in quality. No response could be obtained from the muscles below the knee. Some weeks later considerable muscular wasting was present, extending as high as the right gluteal muscles. In six months she could walk without a stick, and eventually recovered, except that the affected muscles remained rather stiff.

In a system predisposed by the effects of previous disease or excesses, and reduced by the exhaustion, excitement, and strain of labor, septic or other infection attacks the nerve centres or fibres, and as a result, according to the degree and extent of the onslaught, neuritis with or without palsy, or myelitis, or intra-cranial inflammation with convulsions, or psychical affections or other brain symptoms, may occur.

A woman, recently a patient at the Polyclinic, a few days after labor, and after an effort to stop the flow of milk, was attacked for the first time with frontal headache, which has persisted with a few intervals of relief for twelve years, during which time she has passed through five other labors, losing two of the children in early infancy. This ache, probably initiated by a mild infectious neuritis, has never left her.

Recently, in the post-mortem room of the Philadelphia Hospital, I was present at an autopsy on a patient from the wards of Dr. E. P. Davis, this case illustrating the other extreme. This woman, three hours before she was brought to the hospital by the police patrol, had been delivered of a child without proper attention and with wretched surroundings. She was suffering from hemorrhage until after admission to the hospital, and was in a state of acute mania. In addition, her limbs, particularly the upper ones, were extremely hyperæsthetic. Her head was drawn backward somewhat to one side, and active contractures were present in the upper limbs, and possibly also in the lower, but these were not closely examined. She had a scattered petechial eruption. She died about thirty-six hours after admission. The symptoms were such as to suggest cerebro-spinal meningitis. The autopsy showed clearly that no meningitis was present. Sections of the brain and cord showed them to be blanched and dry. Doubtless this was a

case of profound toxæmia, associated with anæmia, caused by the great loss of blood. The patient's blood was in the puerperal condition, the surroundings of her labor were depressing, and she was of the class of those who so often suffer from the ravages of alcoholism or syphilis.

NEURITIS AND PARESIS ASSOCIATED WITH PHLEGMASIA ALBA DOLENS.

Neuritis and the partial paralyses of phlebitis, and phlegmasia alba dolens, can be conveniently discussed as a separate class, although perhaps they might be included under other heads given.

Patients suffering from phlegmasia alba dolens are, of course, more or less helpless and paretic during the progress of the affection; but, in addition, it sometimes leaves forms of paralysis and pseudo-paralysis. Doubtless a true neuritis is often present in phlegmasia, either as a result of the spread of inflammation by contiguity, or because of pressure and interference with the nerve trunks. A form of atheromatous neuritis is now recognized, and has been described by Gowers as a variety of the senile form of multiple neuritis; and it is held that in this disease the nerves of the limbs have been extensively damaged through the obliterating arteritis, necrotic inflammatory processes going on in the parts of the nerves supplied by the affected vessels, either arteries or veins. These processes may or may not result in occlusions, and are frequently accompanied by a true neuritis, which is the origin of most of the pain. In phlegmasia, likewise, a true neuritis may set up; and this may in part persist after other symptoms and conditions have subsided, or nerve degeneration may take place as the result of the neuritis, or of the pressure exercised on the nerves by the swollen and indurated tissues. Even the gangrene, in which phlegmasia sometimes terminates, has been regarded as in part at least neuro-trophic rather than altogether due to interference with the circulation. Finally, a patient suffering from phlegmasia may at the same time, or as a sequel of the affection, develop a septic, pyæmic, or infectious myelitis of the transverse or some other variety, which will give rise to marked and, it

may be, incurable paraplegia. Winckel quotes Mauriceau, Boer, Casper, and Gittermann as reporting a greater or less degree of paralysis of the affected limb as left over by phlegmasia. Dr. Anna M. Fullerton has furnished me with some notes of several cases of this character, patients who have been confined at their own homes without proper antiseptic surroundings and attendants, and have come to the hospital for treatment. She believes that inflammatory and septic complications are responsible for this class of cases; in them she has found extensive lesions, often of the soft parts, due to injuries occurring during birth, which have left open surfaces for the absorption of the poison. The following are notes from cases furnished by Dr. Fullerton:

Case V. *Pyæmia, phlebitis, anæsthesia, and pseudo-paralysis.*—A. H., married, aged twenty-seven years, pelvis generally contracted, was delivered of her second child six weeks before admission to the Woman's Hospital. The labor was said to have been easy. The condition of the patient during her pregnancy had been poor. The drainage of the house and the vicinity was poor, and she thought she had chronic malaria. She had suffered some time previous to her marriage with uterine trouble, and during her pregnancy had been under the constant care of physicians to ward off threatened abortion. Her second labor was followed by an attack of sepsis. When able to be moved she was brought to the hospital. Upon examination an abscess was found over the right sacro-sciatic foramen, and a bedsore on the buttock of the same side. A left crural phlebitis existed with stiffness of the knee, and an œdematous and anæsthetic condition of the leg. The uterus was enlarged and tender, and found imbedded in a mass of pelvic exudate. She was placed upon supporting treatment. The limb was first treated by elevation and application of belladonna and iodine ointment. The pelvic condition was treated by douches and hot packs. The abscesses were evacuated and washed out with antiseptic solutions. Later the limb was treated with massage and electricity. She returned to her home much better six weeks after admission. She was able to walk, but the limb was still weak, and continues so up to the present time, January, 1893. The pelvic exudate has been absorbed. The uterus is movable, but a left salpingitis exists with distention of the tube, for which laparotomy has been advised.

Case VI. *Phlegmasia and neuritis.*—D. R., single, aged twenty-two years, was confined at the Woman's Homœopathic Hospital early in October, 1892, with her first child. Labor was said to have been long, but not instrumental, and was followed by phlegmasia dolens affecting both limbs, the left being first affected and greatly swollen. She was admitted to the Woman's Hospital January 8, 1893, for severe pain in the left foot, affecting

especially the great toe and the two next to it, and extending into the ball of the foot; some pain was in the entire limb, with imperfect use of it. She had enlarged inguinal, axillary, and cervical glands; she was and still is pale and anæmic, remaining under treatment, which has been supporting, with elevation and rest of the limb, and application of belladonna ointment. Considerable improvement has taken place in two weeks. Pelvic examination showed a laceration of the cervix, extending to the vault of the left side of the uterus, which is movable and larger than usual.

Case VII. *Pyæmia with ankylosis, atrophy, and pseudo-paralysis.*—M. T., aged twenty-seven years, married, entered the hospital, October 30, 1891, threatened with premature labor at seven months. It was discovered that she had made attempts to induce labor for some time by means of vaginal injections. Her general condition on admission was very poor; she was pale, anæmic, and apparently half-starved, with albumin in the urine. Notwithstanding the efforts made to arrest the threatened miscarriage, labor occurred on November 5th, being preceded by a chill and rise in temperature. After the delivery her condition remained fair up to about the eleventh day, when again there was a chill and rise in temperature. She had complained of some pain in the right limb since delivery. A synovitis of the right knee with considerable swelling, and complaint of pain and stiffness in the limb, developed. Later suppuration of the knee occurred, necessitating evacuation and drainage. Other metastatic abscesses occurred. The knee became ankylosed; the muscles of the limb greatly wasted. After a long course of supporting treatment, with massage and electricity applied to the limb, she recovered so that she was able to walk without pain, but with a slight limp. She was discharged March 21, 1892.

Case VIII. *Pyæmia with paralysis, atrophy, and anæsthesia.*—M. F., aged twenty-six years, married, flat pelvis, was confined at her home with her first child. The labor was long and difficult, requiring instrumental delivery. The child was stillborn. There were extensive lacerations of the soft parts. The patient was kept to her bed for sixteen weeks with septic fever. The physician who attended her spoke of the position as having been a posterior variety of the occiput. The patient was admitted to the Woman's Hospital, February 1, 1890, with the left limb paralyzed, wasting of the muscles, and some loss of sensation. Pelvic examination showed the existence of bilateral laceration of the cervix uteri to the vaginal vault. The pelvis was filled with an inflammatory exudate, fixing the uterus, which was large and tender. She was placed under treatment, with douches and hot packs for the pelvic condition, and daily faradization of the paralyzed limb, with general restorative treatment. She was discharged March 16th, greatly improved, but with locomotion still imperfect. She returned to the hospital September 22, 1891, for second delivery, although she had been advised in a similar condition to have premature labor induced. The os was found to be fully dilated; the fœtal head

showed no attempt at fixation. Version was performed, and delivery was affected without any injury to the mother. The fœtus was born in the second stage of asphyxia, and could not be resuscitated. This puerperium was perfectly normal. The patient had entirely recovered from her paralysis.

PUERPERAL MYELITIS DUE TO SEPTIC OR OTHER INFECTION.

No reason exists why myelitis, or even meningitis or cerebritis as well as neuritis, may not occur as a result of infection during the puerperium. My own notes only include one case of myelitis. In this case the occurrence of two attacks at intervals of several years, and evidently myelitic in character, is interesting in connection not only with the history of alcoholism, but also with the view that disseminated myelitic foci in the great majority of cases are septic and infectious. The following are the notes of this case.

Case IX. *Transverse myelitis with persistent paraplegia; four years later, bulbar myelitis; history of alcoholism.*—H. L., aged forty-one years, white, a patient in the nervous wards of the Philadelphia Hospital, was married when twenty-two years of age and had seven children. One of her sisters died of phthisis. She denied specific history, but admitted having used alcohol to excess. Her menstruation had been painful up to her last labor, and since then had not occurred. At the time of or just after her last labor, nearly five years before the time she was studied, she became paralyzed in both legs, but suffered no pain. From this time also she was unable to see distinctly as before, and she thinks that her eyeballs began to be a little more prominent. Her condition remained without much change for nearly four years. In a short time she was able to sit up, but she never, since the attack, has been able to walk. A few months since, while sitting in her chair, her speech suddenly became thick and she lost the power in both arms, the paralysis of her legs, which had improved somewhat, at the same time becoming worse.

At present she is unable to stand; she can flex and extend her legs in a feeble manner while sitting or lying down, but has no control over the movements of her feet. Her legs are lean, but not atrophied. Her arms have nearly regained their usual power. She has no loss or perversion of sensation, although she says that at one time her limbs were completely anæsthetic. No pain or hyperæsthesia is present. Bladder and bowels are not paralyzed. Toe-jerk, ankle clonus, front tap, with exaggerated knee-jerks and muscle-jerks, are all present. In short, the paralysis and other phenomena in the lower limbs are such as we would expect from a transverse lesion of the dorsal cord. As results of her last attack, lips and tongue

are paretic, she has slight palatal paralysis, and is unable to phonate well. She also apparently has some exophthalmus, which she believes has been present several years.

The best explanation of this case would seem to be that by infection, or in some unknown way, an attack of myelitis was set up during or after labor, this spending its chief force upon the dorsal cord, although at first, as in other cases, the whole or a large portion of the cord may have been involved in the inflammation. Her recent attack was probably pathologically of a similar character—a central bulbar myelitis. The alcoholic history is of interest, as chronic alcoholism, like syphilis or tuberculosis, would predispose a puerperal woman to such an attack. Neuritis, either diffused or multiple, was not present. The blindness of one eye and the protrusion of the eyeballs may be explained on the theory of the occurrence of foci of inflammation in some of the centres at the base of the brain or in the ganglia of the sympathetic.

Handford, in the paper cited, reports a third case of which he expresses doubt as to its neuritic character, or at least of its being one of simple neuritis. Its onset after confinement, however, was clear, and the case has some symptoms pointing toward neuritis. The following are Handford's notes:

"In this case the diagnosis is difficult, but it lies between tabes dorsalis, selerosis with patches in the crura and in the lumbar enlargement of the cord, and multiple neuritis. There certainly is ataxy; but against the affection being tabes are the facts that she has never suffered from lightning pains or gastric crises; the pupils are dilated, there is no atrophy of the optic discs; the arms were affected as early and as severely as the legs; there is distinct loss of power, and now distinct muscular wasting. There was no evidence of syphilis, and her children are very healthy-looking. E. S., aged thirty-one years; married six years, three children. She suckled the last a year and a half. Difficulty in walking and a weakness in the arms came on after her confinement two years ago. In the house she used to have to assist herself by the furniture, and in the street walk along by the wall. On admission, in July, 1888, she could not stand, and could only feed herself with one hand with a spoon. The legs were very feeble, and occasionally jumped and became drawn up without the patient's knowledge. The knee jerks were totally absent, as were also all the superficial reflexes. Cutaneous sensibility was much diminished in the arms and legs. There was double ptosis and divergent strabismus with widely dilated pupils, moving to neither light nor

accommodation—a complete paralysis of both nerves. Sight was good in each eye and the disks were normal. There was incoördination and loss of muscular sense. She soon went home, and I have been able to keep her under observation up to March, 1891. The duration of the illness, is therefore, five years. There is no appreciable alteration except that the voluntary movements of the arms are more irregular, and the left is much weaker than the right. There is no rigidity of the legs. The thighs are well nourished, but there is considerable wasting of the muscles below the knee on the left side. She cannot stand, but spends her time entirely in bed; eats, sleeps well, and is free from pain."

A close reading of this case would seem to indicate that in all probability the patient suffered from a form of diffused myelitis, the inflammation attacking, beside the cord proper, the basal nuclei. The active sensory symptoms of neuritis were absent in the case, although diminished sensibility was present.

To hysterical and reflex paralyses I shall only refer, as the chief purpose of the paper is to direct attention to the organic forms of post-puerperal affections, peripheral and spinal. Any form of hysterical paralysis may occur, in one predisposed, under any sufficient exciting cause, and certainly pregnancy or the puerperium is sufficient for the production of any functional nervous affection. A few cases with hemi-anæsthesia, evidently hysterical, have been put on record as occurring during gestation or in the puerperal period, as, for instance, by Churchill,[1] who is quoted by Lloyd.

The reflex theory has been used for these as for other cases to escape difficulties which the industry or the ability of the physician has not been able to overcome. Instead of paralysis being due to some vague form of reflex action dependent upon an abnormal excitement exhausting the spinal centres, it is more reasonable to assume that, at least, in not a few of these cases actual transmission of the inflammatory process by ascent to the cord occurs; or that a septic or infectious agent in the blood poisons the centres and nerve tracts, and gives rise to a true toxic palsy; although in extreme instances a puerperal excitement may be so sudden and so great as to overwhelm the centres to which it is transmitted, and to produce by exhaustion temporary paralysis. Cazeaux and Tarnier

[1] Churchill: Dublin Quart. Journ. Med. Sci., vol. xvii., and op. cit.

quote Jaccoud in explanation of the reflex origin of the paralysis of gestation and childbed as of other paralyses. Many such paralyses are said to be occasioned by exhaustion of the nerve centres. A long-continued excitement transmitted to the spinal cord by the uterine nerves exhausts the excitability of the spinal centres, and these exhausted centres are no longer able to transmit the motor impulses to the brain, hence paralysis results.

As many of the cases are of septic or infectious origin, and as this infection may in some instances be introduced from without, the importance of extreme cleanliness and antiseptic measures is emphasized by these as well as by other accidents and consequences of labor. Occasionally, as in times of epidemics, influences from without, and not in any way connected with the labor, may be efficient to cause a multiple neuritis or myelitis, and these cannot be guarded against except by attention to the general health of the patient.

In treatment as well as in diagnosis it is of the utmost importance to consider the neuritis, intra-pelvic or extra-pelvic, which is present in these cases. When sure that neuritis is present, even if it is only of a moderate degree, but particularly when severe, the patient should be kept in bed, or, if on her feet, should be put back to bed. Rest alone will do much for these cases. Even where a persistent and perhaps permanent peroneal paralysis has been left, it is not necessary that the patient should suffer indefinitely with nerve inflammation, and the degree of improvement of the paralysis which will take place cannot be determined until the neuritis has been subdued ; nor can thorough local treatment with electricity, massage, and Swedish movements be carried out while the limb is in a painful state. In cases of the second class which have been here discussed—those of neuritis with pseudo-palsy and affections of the intra-pelvic viscera—treatment of the neuritis by rest and other measures should go hand-in-hand with the gynecological and general measures. These patients will not be firm on their limbs as long as their pelvic nerves are in an inflamed condition, and as long as these inflamed nerves are nagged and worried by exudates or by enlarged and displaced organs.

By means of sand-bags between, and to each side of the legs and

to the feet, the limbs may be kept in a state of quietude as nearly absolute as is possible. Hot douches, or douches of hot and cold water rapidly alternated, can be applied for a few minutes at a time, twice or three times daily. If necessary, opiates may be used, but when possible they should be avoided. Even in the absence of syphilitic history, I commonly resort to inunctions of mercury until a slight constitutional effect is produced. A good ointment is one containing a mixture of mercurial ointment and lanolin, to which a measured amount of sulphate of atropine can be added. An ounce of the ointment may be divided into thirty-two parts, so that each part will contain about $\frac{1}{160}$ to $\frac{1}{130}$ of a grain of atropine; then each of these parts of ointment can be wrapped in paraffine paper, and one to four can be used daily. A good time to use the inunction is immediately after the douches.

Between the times of these applications it is desirable, if possible, to keep the limbs in a mild perspiration, which may be accomplished with strips of rubber dam, such as is used by dentists and surgeons; wrapping the limbs loosely with strips or layers of this fastened with tapes.

Internally in the acute and subacute stages, salicylate of sodium, salicin or salol, in full or fair doses, will sometimes be found very efficient; and phenacetin or antipyrin can sometimes be advantageously combined with these remedies. When the affection has become more chronic, iodide of sodium or potassium, or hydriodic acid, can be added or can be substituted for the earlier treatment.

It may be necessary to use moderate doses of tonics, such as strychinine or quinine, from the first. Weak galvanic currents can be used before the pain has entirely left, as can also massage and Swedish movements; but the active electrical and manipulative treatment is rather for the paralysis which results from the nerve-crushing and neuritis than for the neuritis itself. Appropriate gynecological treatment should be thoroughly pursued.

DISCUSSION.

Dr. Barton Cooke Hirst: I have been greatly instructed and entertained by Dr. Mills' valuable study of this interesting subject, and have been most interested in the mechanical explanation given to account for paralysis after labor. This is a very rare occurrence in the practice of the obstetrician, and this is the reason that the subject is so inadequately treated in the text-books. I have had a rather large experience in contracted pelves, difficult labors, forceps deliveries, and head impaction for periods exceeding two days; but I think that in only a single instance have I seen well-marked paralysis of the limbs as a result. I find also that obstetricians in Europe have seen few cases of this kind. Winckel in 20,000 labors has seen but a single instance of absolute paralysis of the leg following labor. I have read of these cases, and know that the trouble has been attributed to direct pressure upon the nerve trunks in prolonged and difficult labors, but I am not able to subscribe unreservedly to this view. We have all heard women complain of numbness during and after labor, and have seen partial failure of power in the limbs for a little while, but it disappears in a few days without treatment. The only case that I have seen in which paralysis persisted was in a woman two weeks after labor, and I promptly sent her for further treatment to a neurologist.

A month or two ago I made a careful dissection of the pelvis, to see why it was that difficult labor with contracted pelvis was not more often followed by these nerve injuries. In this dissection I was impressed with the fact that the nerves are so well protected by their situation that it is little wonder they are not often pressed upon. The ordinary pelvis has a cordiform entrance, and the nerves are hidden within the bay, so that no mechanical pressure is possible. If the pelvis is contracted the head assumes a transverse position, and the jutting forward of the promontory keeps it from the nerves. In studying these specimens I was impressed with the belief that we might meet with the accident in those rare cases in which the pelvis has an elliptical shape. This was formerly considered to be the ordinary form of the pelvic inlet, but it is not so. I do not know of a single specimen in my large collection which shows it. In such a pelvis I can conceive that the nerves might be easily pressed upon.

As regards the other kinds of palsy, there is no difference of opinion. We have all seen nerve-irritation and inflammation in phlegmasia. I have had seven such cases. It is well known that plastic exudates can press upon these nerves, and the inflammation extend directly to the neurilemma. The idea of direct pressure of the head upon the nerves has seemed to me to be unwarranted in the majority of instances, and the only explanation that I can offer for those rare cases, in which pressure alone could account for

the paralysis, is that of abnormality in the shape rather than in size of the pelvis.

Dr. Wharton Sinkler: I was especially interested in the point made that some of these cases of neuritis following labor are of infectious origin. In studying this subject I had come to the conclusion that the majority of cases of neuritis following labor were the result of some septic poisoning. I have met with only a few cases of neuritis caused by childbirth. The most striking case was one seen in consultation with Dr. Parish some six weeks after delivery.

The patient was about twenty-six years old. Labor occurred at full term—a large child presented by the vertex. After about eighteen hours of labor Dr. Parish applied the forceps (Tarnier's) above the superior strait. The delivery was effected in about an hour after the forceps was applied, and without the exercise of more than a moderate amount of force. The difficulty of delivery was due to the existence of a small pelvis and a large firm head. Motor paralysis was apparent as soon as the patient recovered from the etherization. At first the paralysis involved the muscles supplied by both the obturator and the sciatic nerves in both lower extremities. At no time was there any rise of temperature above 99° F., nor were there any other evidences of sepsis. Dr Parish believed that the paralysis was due to compression of the nerves by the head during its extraction. There was marked loss of sensation from the termination of labor.

When I saw the patient there was a general neuritis of the lower extremities. There was tenderness over the nerve-trunks, hyperæsthesia, and a considerable amount of muscular paralysis. The flexors of the feet were palsied, causing the foot-drop which is so characteristic of multiple neuritis. The knee-jerks were diminished, but the muscles all responded to the faradic current.

Her next labor was more difficult than the first one—the family having declined the induction of premature labor, and a consultant coinciding with the family in the opinion that such a procedure was unnecessary; the second labor terminated fatally to the child, as did the first. The mother suffered from contusions of the pelvic viscera, but escaped from any manifestations of disturbance of the motor or sensory functions of the nerves which pass through the pelvis. Her recovery from this labor was also complete.

I have also seen two cases of neuritis of the upper extremity following labor. These must also have been of infectious origin. Of course, we frequently meet with cases of sciatic neuritis and inflammation of the anterior crural nerve as the result of pressure, but these cases usually recover in a short time.

Dr. Francis X. Dercum: Dr. Hirst has remarked that in the experience of obstetricians these cases are rare. I think that it can also be said that from the standpoint of the neurologist they are not by any means frequent. Taking, together, my service at the Orthopædic Hospital, that at

the University Hospital, that at the Jefferson Hospital, and that at the Philadelphia Hospital, I have seen but three cases in which nerve-injury seemed to follow labor. Further, one of these cases could not properly be ascribed to labor. The history was that of a rapid on-coming paraplegia following labor; at the same time there was a specific history in the case, and the woman made a good recovery under specific treatment.

A second case was seen outside, in a woman who had had a severe instrumental labor. There was intense neuritis, which seemed to take its origin in the sacral plexus and to involve all the nerves of one limb, with the consequent loss of power, loss of knee-jerk, pain and foot-drop, so well described just now by Dr. Sinkler.

I have seen a third case following labor. This was also an instrumental delivery. There was foot-drop, but no history of pain in the pelvis and no tenderness over any of the nerve-trunks except, perhaps, the anterior tibial. The nurse had held the limb for some time in a constrained position, and it occurred to me that possibly the case was one of pressure palsy of the anterior tibial nerve. Anterior tibial palsies are rare, but I have seen several cases.

To-day I saw for Dr. Davis another case of foot-drop following labor. I did not detect any tenderness over the nerve trunk, and there had been prompt recovery from the foot-drop. There is possibly a little tenderness over the dorsal nerve of the foot.

This embraces the sum total of my experience with these curious nerve-injuries. While they do occur, I think there can be no doubt that their occurrence is very infrequent, if not rare.

Dr. James Hendrie Lloyd: A few years ago I had occasion to study this subject by dissection, in preparing a short paper for Hirst's *System of Obstetrics*. I also studied the literature. I was then impressed with the fact that the literature was of very little value and also with the fact that, as Dr. Hirst has said, pressure does not explain these cases, except, perhaps, in some forms of instrumental labor, where the sacral plexus or sciatic nerve is involved. My belief is that in the majority of genuine cases we have to do with an infectious neuritis or myelitis, which does not differ from the same disease under other circumstances except, perhaps, in its distribution.

Dr. Mills made a brief allusion to hysteria. I think that this is an important phase of the question, and that hysteria is a possible explanation of some of these cases. I have known, by report, of a number of cases of women who, after labor, have had for a long time various obscure paralytic affections. One case had hemiplegia and hemi-anæsthesia for a long time. From the reports, I am led to believe that these were cases of hysteria. In some cases there was blindness. I think that this was probably due to hysteria, perhaps aggravated by excessive doses of quinine. A French observer has written an elaborate paper on aphasia following labor. A certain number

of his cases were due to hysteria. This is a vital question for differentiation, and important for the welfare of the patient.

Dr. E. P. Davis: The case to which Dr. Dercum made allusion is that of a primipara who bore a full-sized male child after an instrumental labor. The position was the second, the occiput to the right. The pelvic floor was lacerated, but was promptly repaired and healed without rise of temperature. The patient presented no hysterical symptoms during convalescence, but as convalescence proceeded the foot-drop became evident. There were disturbances of sensation and of motility, first in both lower extremities, and then in one. The patient has, however, made a very satisfactory recovery, without other treatment than rest in bed. All obstetricians and gynecologists at times see cases of neuritis of the infective variety, but distinctly obstetrical paralysis of the lower extremity, caused by pressure during labor, is of great rarity.

SYMPHYSIOTOMY VERSUS ITS SUBSTITUTES; WITH THE REPORT OF A CASE OF SYMPHYSIOTOMY.

By CHARLES P. NOBLE, M.D.,
SURGEON-IN-CHIEF, KENSINGTON HOSPITAL FOR WOMEN, PHILADELPHIA.

[Read February 1, 1893.]

The revival of symphysiotomy is due to the genius of Morisani, of Naples; its introduction into the United States, to our distinguished Fellow, Dr. Robert P. Harris. Now that the results of symphysiotomy are known, as accomplished by the operation done under modern aseptic methods, the wonder is that the profession could have been blinded by the prejudices of the past, arising out of the comparative failure of the operation when done without regard to a knowledge of scientific obstetrics or surgery.

The results obtained by Italian operators under the influence of Morisani's teachings took the world by surprise. The fact that the work was being done was well known, but the profession was supercilious and refused to estimate it in a scientific spirit. The old, prejudiced belief that symphysiotomy was necessarily a failure because it did not permit of an increase in the conjugate diameter of the pelvis and because it jeopardized the integrity of the pelvic symphyses, was too firmly grounded to be easily set aside, and it required the demonstration of the safe delivery of thirty women, in Italy, having contracted pelves of marked type, before any but Italian surgeons were willing to perform the operation.

The old objections to the operation were: 1. That in con-

tracted pelves, as a rule, the antero-posterior diameter is the shortened diameter; and that this diameter is scarcely, if at all, increased by the operation. 2. That there is great danger of rupture of the sacro-iliac synchondroses. 3. That the symphysis pubis would not unite properly; and hence that the powers of locomotion in women subjected to the operation would be greatly lessened. Experience has shown each of these objections to be groundless or much overstated. Undoubtedly the oblique and transverse diameters of the pelvis are increased more than the antero-posterior, but this diameter also is increased. When the pelvic bones are separated from two to two and a half inches, enough room is afforded for the rounded head (perhaps the parietal protuberance) to project into the opening, thus increasing the working space at least half an inch. This increase in space, together with the increase in the oblique diameters, affords the necessary room for delivery, except in cases of very marked deformity.

The objection that the sacro-iliac joints may be ruptured has not been borne out by experience. Nevertheless it should not be forgotten that at times the pelvic joints are ruptured in cases of difficult labor in which symphysiotomy has not been done. This should admonish surgeons to be careful in recommending the operation in cases of marked deformity, especially if the head of the child seems large and well ossified.

The final objection, that the divided symphysis would not unite, is likewise not well founded, as experience has demonstrated that it unites with pleasing uniformity and facility.

Since January 8, 1886, there have been fifty-seven symphysiotomies, and all the women have recovered, except one. This woman (case of Martino, September 22, 1886) had been in labor several days when operated upon, and died of metroperitonitis, due to infection of the birth-canal, and not to the operation. This fact justifies the conclusion that in proper cases when done early, and after the antiseptic method, symphysiotomy has no mortality. This is true, however, only under the conditions laid down. The history of the operation

admonishes us that when done on improper cases, and without asepsis, its mortality is high.

The complications following the operation have been urethral and vesical fistulæ in a few cases. According to Morisani, these were due to a faulty technique. Not having had the opportunity myself to study the recorded cases of symphysiotomy, to discover accidents resulting from it, I wrote to Dr. Harris, who has recently studied the literature in the preparation of his paper on the subject (*Trans. Amer. Gynecological Society*, 1892). He writes me that since the perfection of the technique, in 1886, there is no case recorded in which the sacro-iliac joints have been injured; no case in which a child has been delivered absolutely dead; and no case in which it was necessary to practise embryotomy to complete the delivery. Even going back to 1866, in 125 symphysiotomies there is but one case recorded of possible injury to the sacro-iliac joint. This was a case of iliac phlegmon, which was by no means necessarily due to joint-injury.

The results to the children delivered by symphysiotomy have been equally good. No child has been born absolutely dead, but six children out of fifty-seven died shortly after birth.

Having thus disposed of the questions heretofore raised concerning the operation, it remains to consider its true field of usefulness and its relation to Cæsarean section, the induction of premature labor, and to embryotomy.

The operation has been done, heretofore, principally in cases of flat pelves, and the measurements hereafter given refer to that deformity. Other varieties of deformity are rare, but fortunately the operation is even better adapted in them to facilitate delivery, excepting cases of Naegele or Robert pelves.

The conjugate diameter in the women heretofore operated upon has varied from 66 mm. ($2\frac{5}{8}$ in.) to 97 mm. ($3\frac{13}{16}$ in.). There is a general agreement among surgeons to limit the field of symphysiotomy in flat pelves to cases having at least $2\frac{3}{4}$ inches in the conjugate diameter. I find myself in agreement with this opinion. My own experience in delivering a child weighing eight pounds and two ounces through a pelvis of $2\frac{3}{4}$

inches has admonished me that less room would entail very great danger of death to the child, and would put more strain on the sacro-iliac joints than would be judicious.

In this field symphysiotomy comes directly into competition with the Cæsarean operation, done for the relative indication. I am convinced of the beneficent nature of the modern Cæsarean operation, when done *secundem artem*, and that it has a very low if not a *nil* mortality in the hands of an expert. Nevertheless, it must be acknowledged that the Cæsarean operation is a more formidable operation than symphysiotomy, and that its possibi lities of morbidity and mortality are undoubtedly greater. Hence I unhesitatingly pronounce in favor of symphysiotomy in this field, and believe that it will soon supplant the Cæsarean operation performed for the relative indication. Aside from its absolute claims for preference, there are several relative advantages which I believe will prove of great service in practice. The practitioner will not have to overcome a popula r prejudice, as is the case with the Cæsarean operation. Symphysiotomy is to the public a new operation; hence the public is prepared to estimate its risks as presented by the profession. This is not true of the Cæsarean operation, for the modern operation has to bear the evil name of the old one, and is believed by the public to be almost necessarily fatal. These considerations will render it more easy to gain consent for the performance of symphysiotomy than it has been for the Cæsarean section. Another advantage will be that the failure of the natural forces, assisted by art, to accomplish delivery, will prove a convincing argument in favor of the necessity for symphysiotomy. And, happily, the operation can then be done with success, whereas, in the case of the Cæsarean operation the favorable time would already have passed by.

Symphysiotomy likewise fills the field heretofore occupied by embryotomy done on the living child. Since the perfection of the technique of the Cæsarean operation, in common with many others, I have denied the justifiability of embryotomy done on the living child. With the addition of symphysiotomy

to scientific obstetrics I believe that the slaughter of the innocents will cease. The old excuse for embryotomy, that it is justifiable to kill the child to save the mother, no longer holds, for both can be saved either by the Cæsarean operation or by symphysiotomy. As I have recently discussed this phase of the subject in a paper read before the Philadelphia County Medical Society (*Amer. Journ. of Obstet.*, 1893) entitled "Cæsarean Section and its Substitutes," I have only to add that I believe the greatest good which will be accomplished by symphysiotomy will be that it will put a stop to the killing of unborn infants. Courageous and expert surgeons could save both mother and child by the Cæsarean operation, but apparently they could not do away with the practice of embryotomy.

Symphysiotomy, also, has been performed in the same classes of pelves in which, heretofore, the induction of premature labor has been practised. Hence these two operations come into competition. This is a phase of the subject which heretofore has attracted no attention. This fact renders it inexpedient to dogmatize at this time. But I believe that symphysiotomy is destined to supplant the induction of premature labor, especially as a hospital operation and in practice among the ignorant and the poor. Embryotomy heretofore has been employed to terminate arrested labors amongst the poor having deformed pelves. Such people, as a rule, employ midwives or physicians who do not devote much study to their cases prior to the onset of labor. As a result, premature labor is seldom induced among this class in private practice. Symphysiotomy will now supplant embryotomy among them.

In hospital practice the choice of operation will depend on the mortality of the mother and the child under the two operations. The advantages appear to be decidedly on the side of symphysiotomy. The maternal mortality from the induction of premature labor is variously stated by authorities. For example, according to Wyder's statement 5 per cent. of the mothers and 50 per cent. of the children die. Winckel states that of children born at from seven and a half to eight months only 33 per cent. are actually kept alive. Under the use of

the incubator at the Leipzig maternity there was an infant mortality of 18 per cent. in the hospital, and at the Paris Maternité there was a mortality of 30 per cent. It is also well known that the mortality of premature infants among the poor is very high in the first year of infancy, so that Winckel's estimate is probably correct.

In my judgment 5 per cent. is an excessive estimate of the maternal mortality of the induction of premature labor. One per cent., or certainly 2 per cent., should cover the mortality in careful hands.

The showing under symphysiotomy is much better. The maternal mortality by zero, and the fœtal mortality is six in fifty-seven, or less than 11 per cent. The chances of the mature child delivered under symphysiotomy to reach maturity are likewise far greater than those of the immature child delivered about the thirty-fourth week of gestation. This is especially true of the children of the poor.

Among the intelligent and well-to-do, premature labor will probably be elected for a time; but if symphysiotomy continues as successful as it promises, it will supplant the induction of labor even in this class of cases. Intelligent people will not care to run the risk of losing their children from prematurity, or, what is worse, having them suffer from hydrocephalus (which is common in such children), and from various neuroses, if a mature child can be delivered with less risk to the mother under symphysiotomy.

The technique of the operation can be considered conveniently in connection with the report of a case of symphysiotomy which follows.

Case I.—Mrs. X., the subject of this report, is a small woman, weighing one hundred pounds. She is thirty years of age, and has had five children, the last of which was delivered by symphysiotomy. A complete history of her five labors would be a fairly full consideration of dystocia due to deformity of the pelvis, together with the obstetric procedures used to complete such unnatural labors. The following are the measurements:

Height, 4 feet, 8 inches. A. S. S., 24 cm.; Cr. Il., 26 cm.; Tr., 29 cm.; D. B. (ext. conj.), 16.5; C. D., 8.5; C. V. (estimated), 7 cm.

The first child, a boy, was born after a labor of 19 hours' duration. De-

livery was accomplished by the forceps, the head being so injured by the vigorous compression and traction employed that the infant died shortly after birth. The child was not weighed. The second child, a girl, was delivered alive, spontaneously, after a hard labor of 14 hours. She was not weighed, but was so tiny that she was not expected to live. It is safe to say that she did not weigh *more* than five pounds. The third child, a girl, was delivered by Dr. Howard A. Kelly by Cæsarean section. She weighed six pounds, fifteen ounces. The fourth child, a girl, I delivered, after the induction of labor at the thirty-sixth week, by a high application of the forceps. The baby weighed five and one-thirty-second pounds. The B. P. of the fœtal head was 7.5 cm.; B. T. 6.5. The labor was extremely difficult, lasting 27½ hours. (Vide *Amer. Journ. of Obstet.*, 1890, p. 418).

I met Dr. Kelly in consultation, and concurred in the opinion that Mrs. X.'s third delivery could be accomplished only by craniotomy or the Cæsarean operation. Dr. Harris also had expressed the same opinion. Mrs. X. and her husband unhesitatingly elected the Cæsarean operation. Even had her medical advisers recommended craniotomy she would have rejected it. This was largely due to the fact that she is a Roman Catholic; but doubtless it was partly owing to the horrible death of a sister (who also had a deformed pelvis) under embryotomy—a combination, I believe, of version, craniotomy, and decapitation. The sister died from loss of blood. The issue of the case demonstrated the wisdom of the advice given, as delivery would clearly have been impossible without diminishing the head of a baby weighing $6\frac{15}{16}$ pounds. This operation met with much unfavorable criticism, part of which was due to ignorance of the exact facts in the case, and to the prejudices arising from so-called conservatism, and part of which was due probably to animosity on the part of critics. After this lapse of time those who were immediately concerned in the case can look back upon this operation with equanimity. Undoubtedly it had much to do with popularizing the Cæsarean operation performed for the relative indication in this country.

Had there been a question as to the available room in this pelvis it would be settled by the fourth labor, in which a premature child, weighing $5\frac{1}{32}$ pounds, was delivered with extreme difficulty. This head was delivered with the bi-temporal diameter engaged, and there was no room to spare. The fit was so tight that I feared it would be impossible to deliver the undiminished head. Yet this fœtal diameter measured but 6.5 cm.[1]

The fifth labor is the subject of this report. Mrs. X. consulted me when seven months pregnant. I found that already the head was so large that it could not be pressed into the pelvis. Two weeks later this disproportion was evidently greater, and a careful palpation of the child made at this time led me to believe that it was large rather than small. Knowing the deformity of the pelvis, and having a lively recollection of the difficulties encountered

[1] For a full consideration of the history of this case, *vide* Amer. Journ. of Obstet., 1890, pp. 237 and 418.

at the preceding delivery, I became convinced that it would be wiser to do symphysiotomy at full time than to induce premature labor. I so advised the woman, but asked her to reserve her decision until she had consulted Dr. Parish, who agreed with me in advising symphysiotomy as against the induction of premature labor. So far as I know, this is the first time in which symphysiotomy has been elected over the induction of premature labor.

I opened the symphysis, and delivered by a high application of the forceps, December 5, 1892, at the Kensington Hospital for Women. The cervix being well dilated after a labor of eleven hours, the patient was put in the lithotomy position for operation. The bowels and bladder had been emptied, the lower abdominal, pubic, and pudendal regions cleaned, shaved, and disinfected, and the vagina douched. The membranes were now ruptured. It was not considered worth while to try forceps delivery before proceeding with the operation, because the head was free at the superior strait, and clearly too large to engage. The operation was commenced by making an incision an inch in length in the median line of the abdomen and terminating at the symphysis pubis. This was carried down to and through the muscular and aponeurotic structures. More room was now afforded by detaching the muscle from the pubic bones by cutting transversely with scissors until the left index finger could be passed behind the pubes. The connective tissue back of the pubes is so loose that the finger encountered no resistance, and was easily passed below the pubic arch. The urethra being now depressed and dragged to one side by a catheter in the hands of an assistant, the Galbiati knife was passed along the finger as a guide, and hooked under the symphysis. This was divided by traction on the knife in an upward and forward direction. Much difficulty was encountered, as apparently the ligamentous structures were not abundant, and the knife became wedged between the bones. The symphysiotomy lasted twenty-six minutes. As there was considerable venous oozing, a sponge was packed behind the symphysis and gauze was laid over the wound.

The forceps was now applied, and the baby, a boy, was delivered after an extremely difficult forceps-labor, lasting thirty-seven minutes. This is explained by the fact that the baby weighed 8½ pounds, and that the head measurements were B. P., 9.5 cm.; B. T., 9 cm.; S. O. B. 9 5 cm. After the symphysis was divided the bones separated one-fourth of an inch. After vigorous traction with the Hodge forceps they separated two inches, and the head became well engaged. The Tarnier forceps was now applied, when the head descended into the cavity of the pelvis, and a separation of 2½ inches was obtained. Two assistants now supported the pelvis by pressing on the trochanter upon either side, and drawing upon the ilium of the opposite side. It was interesting to observe that the labor followed the mechanism of the flat pelvis. The head descended semi-flexed, with the occiput to the right ilium and the sinciput to the left ilium, until it began to distend the pelvic floor, when flexion and internal rotation took place. The child was delivered

asphyxiated, and required careful attention for ten minutes before it was resuscitated. The use of suspension, head downward, dipping in warm and sprinkling with cold water finally brought it around. The placenta was now delivered, and the vagina and the uterus were douched with sublimate solution, 1 : 2000. The sponge was removed from the operation-wound and the hemorrhage found to be controlled. Several silkworm-gut sutures were passed through the skin and muscle in each lip of the wound, which came nicely together. In the meantime the pelvic bones were pressed together by the assistants. A dressing of aristol and gauze was applied over the wound, over this rubber adhesive plaster, and finally a stout binder coming well below the trochanters. The legs also were tied together for some days. The patient was put to bed without shock.

In performing the operation I had the advantage of the counsel of Drs. Harris and Parish, and Drs. Boyd and Parish kindly took charge of the baby. The operation was witnessed by a number of physicians, among others the venerable Dr. J. G. Allen.

The after-history was absolutely uneventful—a normal puerperium. Tender and abraded nipples interfered with the baby's nursing, which prevented him from thriving for a time ; but at the present writing he is fat and well. The extreme asphyxia present at birth probably influenced his nutrition for some weeks.

It was interesting to observe the entire absence of pain in the divided symphysis. I had expected considerable pain from the stretching of tissues, especially below the pubic arch ; but the patient expressed herself as being perfectly comfortable.

The after-management of the case was somewhat troublesome, owing to the necessity of using a wide binder to preserve immobility of the pelvic bones. The vulva was washed twice daily with sublimate solution, and well dusted with boric acid. The vagina was douched on alternate days, more especially to assist in the rather troublesome task of keeping the parts clean. This was troublesome, because it was thought best to keep the legs together.

As a matter of convenience, I used two binders. The one next the skin was of two thicknesses of heavy muslin, made with "three tails." The outer one was of canvas. The bandages were secured with safety-pins, and finally, when the muslin bandage was dispensed with, straps and buckles were attached to the canvas bandage.

In cleaning the patient the outer binder was unfastened, and also the *lower* tail of the muslin binder. The legs were then separated just enough to permit cleansing the vulva. About every third day it became necessary to apply a clean muslin binder. The patient was rolled on her side, the legs being in apposition, and the pelvis supported, when the soiled binder was removed, and the opportunity embraced to wash the region of the buttocks. A clean binder was then placed in position, the pelvis well supported, and the patient rolled on her back, when the two binders were fastened. In this

way it was possible to keep the patient clean, and at the same time keep the pelvis immobile.

Union of the joint was apparently complete in two weeks. The sutures were removed in six days. Wishing to run no risks of poor union, the patient was kept in bed four weeks, then sent home, and confined to bed for another week. Shortly afterward she was doing the housework for a family of six.

DISCUSSION.

Dr. R. P. Harris: Dr. Noble's patient came under my observation in May, 1888, when I saw her in consultation with Dr. H. A. Kelly at her own house, and recommended that she should go to the Kensington Hospital for women, and there be delivered by the Cæsarean operation, which was performed a few days later. When she was pregnant for the fifth time I had become convinced of the value of symphysiotomy, and thought it adapted to her case, because of the form and size of her pelvis, and recommended that it should be selected as a preparation for delivery.

Unfortunately for the facility of its extraction, the fœtus proved to be a male, and much larger than any of her former children, so that it made a very narrow escape for its life, being deeply cyanosed and difficult of resuscitation. It, however, thrived well during its mother's recovery, which was free from any pelvic pain or uneasiness. No convalescence after parturition could have been more devoid of symptoms indicative of the enlargement to which her pelvis had been subjected; and when I examined her on the twenty-second day I found pubic union firm and solid.

Dr. Noble's case is one of the most interesting on record, because of its historic character, as symphysiotomy was brought into contrast with the improved Cæsarean section and with delivery under induced labor in the same subject, and all with entire success to mother and child. I know of no parallel case in my obstetric readings, and of no evidence in contrast, that is more to the credit of the recently introduced method of delivery.

Under an improved technique and proper antiseptic precautions, the operation of Sigault has been robbed of all traumatic dangers, and would appear to be devoid of any risk, when regarded from a surgical aspect. Deaths have taken place since the operation was perfected by Prof. Morisani, and no doubt will continue to take place, when women are operated upon *in extremis;* just as deaths follow after delivery by the forceps or in cases in which craniotomy has been performed. What we contend is that the prior condition of a parturient woman is not rendered the more likely to prove fatal by reason of the section and opening of her symphysis to aid delivery.

During the past seven years, four women out of seventy have died, in whose

cases delivery was accomplished with the aid of symphysiotomy: one in Naples in 1890; one each in Helsingfors, Russia, and in Paris, in 1892, and one in the United States this year. The Neapolitan woman was in labor for several days when taken to the hospital; the shoulder presented and the cord prolapsed; delivery was accomplished by version, and death took place in twelve days from metro-peritonitis. The Helsingfors patient died of heart-disease and other organic complications on the second day. She was forty years old, and in her ninth pregnancy. The Paris case had been in labor three days with her fourth child; the forceps had been applied three times at her own home, and four times in hospital; one of the last, antero-posteriorly. She died the day after delivery, and a small perforating laceration was found leading from the uterine cavity into Douglas's cul-de-sac. The fourth death was in a woman almost moribund, having a pulse of 150. The children were lost, except in the cardiac case and the fourth that died. Certainly death could not be ascribed to symphysiotomy in any of these cases.

Since September 29, 1892, this operation has been performed ten times in the United States and once in Canada, saving ten women and eight children. One child died in twenty-four hours, from the effect of long-continued pressure upon its head in the lower pelvis, and one in three days, from meningeal hemorrhage; the third has not been reported. The American operations have been performed, in chronological order, by Prof. Charles Jewett, of Brooklyn; Prof. Barton Cooke Hirst, of Philadelphia; Prof. Anna E. Broomall, of Philadelphia; Prof. Edwin J. Michael, of Baltimore; Dr. Charles P. Noble, of Philadelphia; Dr. J. A. Springle, of Montreal; Dr. Harry McKennan, of Paris, Illinois; Prof Henry J. Garrigues, of New York; Dr. John Milton Duff, of Pittsburg; Prof. William T. Lusk, and Prof. Henry C. Coe, of New York. In Dr. McKennan's operation, not having a sufficiently strong bistoury, the symphysis was divided by means of a narrow metacarpal saw, guarded by the index finger.

We may arrange the history of symphysiotomy into three periods, viz.: 1777 to 1858; 1866 to 1886, and 1886 onward. In the first period there were over 100 operations; in the second there were 73; and in the third, as far as heard of, there have been 70, of which 36 were in the year 1892, divided as follows: In France, 13; in the United States, 7; Italy, 6; Germany, 5; Russia, 2; Canada, 1; Ireland, 1, and Austria, 1. As Italy had 12 in 1891, it is probable that there were as many as 40 in 1892, as against 12 in 1891.

Some men cannot comprehend my statistics, and are led to believe that *some* of the unfavorable cases must have been hidden away from those who aided me in their collection, in order that the improvement in results should appear so remarkable. We have only to state that a large proportion of the reports were copied from hospital records, and that many other statements came through correspondence. An examination into the diminished mortality of ovariotomy, the Cæsarean section and abdominal hysterectomy will

show what marvellous changes have been effected within a few years in the reduction of the death-rate under antiseptic management.

It was at one time thought that a Porro-Cæsarean section, with the stump treated intra-peritoneally, was almost equivalent to a sentence of death, because only one woman was saved out of the first eleven, and yet 25 operations in order, covering the years 1888, 1889, 1890, 1891, proved fatal to only three women. This week brought me a letter from Prof. Leopold, of Dresden, stating that he had performed the Porro operation eight times without the the death of a mother, and that 42 improved Cæsarean sections in the Frauenklinik had cost the lives of but four women.

The results of obstetric surgery in the hands of very skilful men are certainly trying the faith of the casual observer, but they are none the less worthy of credit. Dr. Hubert Riedinger, of Brünn, Austria, was an early operator after the Porro method, having commenced in 1878; and his records show that he has performed 15 sections, saving all of the women and losing but one child.

The late Karl Braun (13), the late August Breisky (8), and Drs. Riedinger (15), and Leopold (8), had collectively 44 Porro operations, and lost but 3 women and 8 children. Such records show the possibility of the operation, but a general hospital report, as of the Maternities of Leipzig, Dresden, and Vienna, is of much more value in the estimation of risks.

If puerperal celio-hysterotomy and celio-hysterectomy have been so largely reduced in their rates of mortality, why should it be regarded as almost incredible that the operation of symphysiotomy, upon women in a *proper condition*, should have no mortality? If what was once regarded as "the murderous operation of *bi-pubiotomy*" has been recently revived in Paris, under Prof. Adolphe Pinard, with the saving of both mother and child, why should we fear the traumatic results of a simple symphysiotomy? In Pinard's case there was a true conjugate of 8.5 c.m., but one sacro-iliac synchondrosis was ankylosed, and the pelvis was oblique, hence the change of method. This operation originated with Prof. Galbiati, of Naples, who performed it on March 30, 1832, upon a dwarf three and a half feet high, having a one-inch conjugate; the child was dead, the woman died in four days of gangrene of the genitalia and vagina. Ten years later, Dr. Ippolito repeated the operation in Naples, with the same result. Prof. Pinard evidently thought that this operation originated with Dr. Farabœuf, of Paris, who proposed it to him, and he recommended that it be called by his name instead of *ischio-pubiotomy*, by which he reported it before the French Academy of Medicine, on January 10, 1893.

Dr. Barton Cooke Hirst: Without doubt this operation has now become firmly fixed among justifiable obstetric procedures. There have already been ten operations in this country, and I shall probably add another to-morrow. I have only one remark to make to-night in regard to this matter. After my own operation, which I have already reported, I had a conversation

with the President, and received from him a very original and brilliant suggestion. Dr. Mitchell asked me if there were not some way of permanently enlarging the pelvis in the operation of symphysiotomy. It seemed to him that by the insertion of ivory pegs, or by other means, the widening of the pelvis could be maintained. This is a brilliant idea, and I had hoped to have had before now the opportunity of trying it upon animals. The proposition is very interesting and deserves careful consideration. The enlargement of the pelvis in symphysiotomy is considerable. A recent German experimenter has examined, post-mortem, the pelves of three or four women within a few days after delivery, and has found that with a separation of the symphysis to seven centimetres, symphysiotomy gave an increase of three centimetres in the oblique and transverse diameters, and of over one centimetre in the antero-posterior diameter. This enlarges the capacity of the pelvis materially. It would be a brilliant achievement if this could be made permanent by the operation, and if by medical skill we could transform the previous anatomic condition of the patient. It seems almost like sacrilege to think of it. It reminds one of adding a cubit to one's stature, but it seems possible, and I hope soon to have an opportunity of testing Dr. Mitchell's suggestion upon pregnant animals.

REPORT OF A CASE OF SYRINGOMYELIA, WITH EXHIBITION OF SECTIONS OF THE SPINAL CORD.[1]

By JAMES HENDRIE LLOYD, A.M., M.D.,
PHYSICIAN TO THE PHILADELPHIA HOSPITAL, TO THE METHODIST EPISCOPAL HOSPITAL, AND TO THE HOME FOR CRIPPLED CHILDREN.

[Read February 1, 1893.]

I AM indebted to the recent monograph of Dr. Bruhl,[2] of Paris, for a brief but clear historical statement of syringomyelia. Ollivier coined the word and applied it, in 1837, to all canals or cavities in the cord, the presence of a canal being considered by him as always pathological. Stilling, however, showed that the central canal of the cord is normal and constant. Then the word hydromyelia was used by Virchow and by Leyden to designate cavities in the cord, which they believed were always expansions of the normal central canal. Hallopeau, in 1869, taught that such cavities were secondary to a myelitis or sclerosis about the natural ependyma. But, in spite of theory and imperfect observation, cavities in the spinal cord occasionally showed themselves entirely distinct from the central canal. Then Simon, in 1875, pointed out that cavities result from the softening and breaking down of a gliomatous tissue; that this tissue is a new, degenerative formation, which may exist even as a glioma, telangiectasic in character; and he proposed to reserve the term "syringomyelia" for these cysts formed in gliomatous tissue and independent of the cen-

[1] We are indebted to the University of Pennsylvania Press for the use of the cuts illustrating this paper.

[2] Contribution à l'Étude de la Syringomyélie, par le Docteur I. Bruhl, Paris, 1890.

tral canal. This distinction is now generally recognized, and syringomyelia is regarded as the product of a true gliomatosis.

Syringomyelia, having been thus recognized anatomically, has now entered fully into its clinical phase. A long series of memoirs, says Bruhl, seek to establish that it has its own proper symptomatology, and that the diagnosis can be made at the bedside. He adds that an autopsy in many cases has verified the exactness of the diagnosis.

These are precisely the conditions that existed in my own case, for the diagnosis was made upon the patient's admission to the hospital, and the autopsy two months later revealed the extensive and interesting cavity in the cord, sections showing which I have the honor to present to you this evening.

I desire first to state briefly the main features of the symptomatology upon which the diagnosis of this affection rests. Following the admirable classification of Charcot,[1] we note two main groups: 1. The *intrinsic* symptoms, or those related to the limited lesion in the central gray substance of the cord. Here we distinguish (*a*) the symptoms of anterior poliomyelitis —progressive muscular atrophy of the type Aran-Duchenne; (*b*) the symptoms of posterior poliomyelitis—anæsthesia to pain and to heat and cold, with preservation of tactile sensibility, and of the muscular sense (this is the well-known dissociation symptom); (*c*) symptoms of central poliomyelitis, a group whose origin is very problematical, including diverse trophic disorders other than those referable to the muscular system. 2. The *extrinsic* symptoms, or those which do not result directly from the gliomatosis in the gray matter, but are frequently associated with the others; they result from the extension of the lesion to the white substance of the cord, and are secondary. They include symptoms of lateral sclerosis, as spastic paresis or paralysis, and those of posterior sclerosis, or tabes. While this classification, like most others, is perhaps artificial in some respects, it is helpful to those who for the first time attempt to master the rather complicated symptom-groups of

[1] Bruhl, op. cit.

this affection. It strikes me as doubtful whether the symptoms of lateral selerosis should be regarded as always secondary, and whether the muscular atrophy is always of the type Aran-Duchenne. In my own case the type, especially on one side, was spastic. It may also be questioned, as, in fact, Charcot does question, whether the trophic lesions are due to the involvement of the mid-region of the gray matter of the cord. It is evident that we must admit varieties or distinct types of the affection, as, in fact, the very nature of such a variable and destructive process suggests. In the main, however, the diagnosis rests upon the recognition of certain groupings of the above-named elements. The most common grouping is that of muscular atrophy, especially in the shoulders and arms, with the peculiar "dissociation" sensory symptom, and with a variety of trophic disorders. My case is as follows:

E. B., white, male, aged thirty-one years, was admitted into the Philadelphia Hospital in July, 1892. His family history was negative. The patient had not used alcohol to excess, and denied syphilis. He said that he had always been strong until the winter of 1888, when, after a severe storm, he had a swelling in his right ankle, which he regarded as rheumatic. He noticed soon afterward that he was obliged to step on his toes, and that his shoes became worn on the front parts of the soles. He was in the hospital for a few weeks in 1889. His condition on admission last July was as follows:

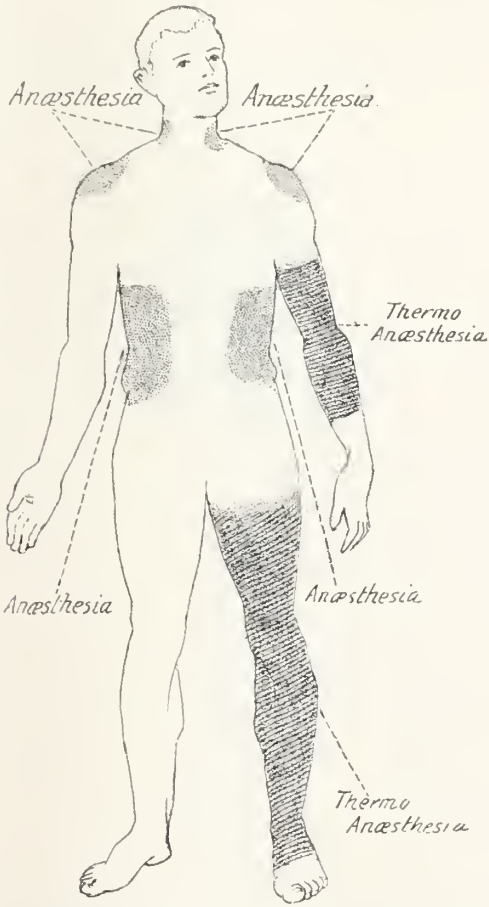
Motor symptoms. The patient had advanced progressive muscular atrophy of the spastic type (the so-called amyotrophic lateral sclerosis). The shoulder muscles, including the deltoid, infra- and supra-spinati and the lower part of the trapezius were much atrophied. The upper part of the trapezius (well called the *ultimum moriens*) was in good condition. The upper-arm muscles, *i. e.*, biceps, triceps, and deeper muscles, were distinctly wasted. Most of these muscles presented a high degree of reflex irritability, and the biceps were spastic. The biceps-jerk was exaggerated, and a very lively reflex was produced by tapping vigorously the back of the wrist while the hand was dropped. The lower-arm muscles also were atrophied and weak. Some of these muscles presented fibrillary contractions. The legs were spastic and contractured. There was no true muscular atrophy or fibrillation in the legs. The knee-jerks were much exaggerated and ankle clonus was present; but this spastic condition, with exaggerated reflexes, was distinctly more marked (both in the arm and leg) on the right side. The muscles did not present the reactions of degeneration. To faradism all the wasted muscles of the chest, shoulder, arms, and forearms reacted normally. The same was

Fig. 1.

A case of syringomyelia.

true of the muscles of the legs. To galvanism the cathodal closure reaction remained greater than the anodal closure reaction in the wasted muscles of both arms. The anodal and cathodal opening reactions were not found. The formula remained normal in the leg muscles. In some wasted muscles

FIG. 2.



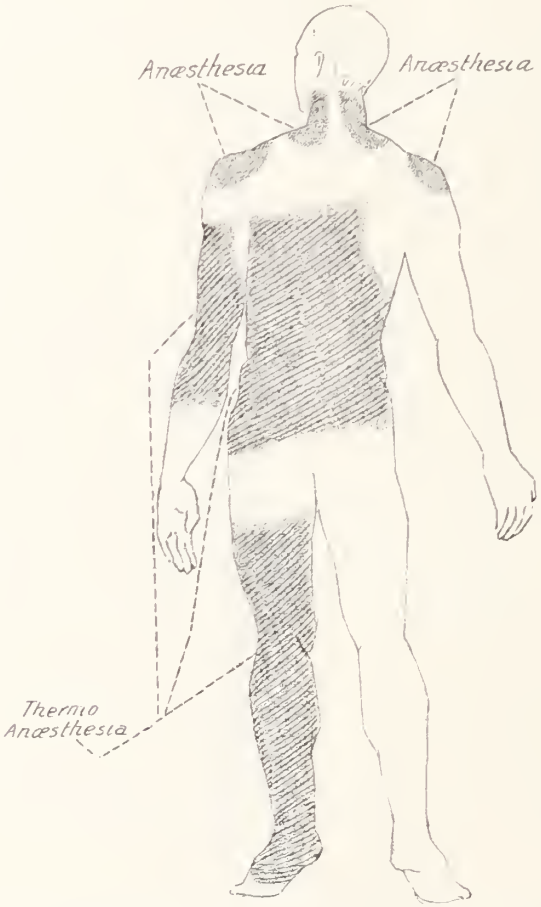
Syringomyelia, showing segmental distribution of sensory symptoms.

of the arms there was a slight modal change. It was thus seen that the case presented the spastic type of progressive muscular atrophy, and the interesting fact was observed that this was much more marked on the right than on the left side.

Sensory symptoms. The patient presented a quite typical picture of the

"dissociation symptom" of syringomyelia. Thermo-anæsthesia, or inability to distinguish heat and cold, was seen, especially in the left arm, both above and below the elbow, and in the whole of the left leg, on both the anterior and posterior aspect; also upon the back (Figs. 2 and 3). Analgesia, or the loss of

Fig. 3.

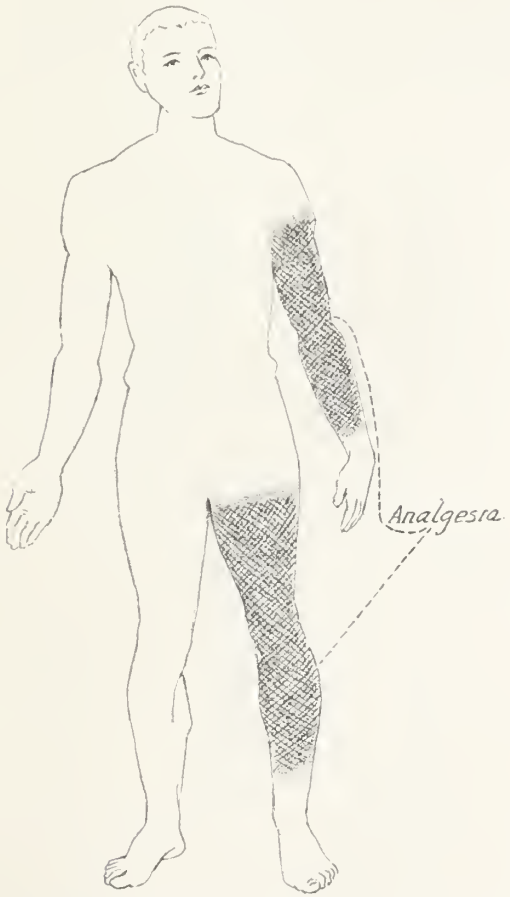


Syringomyelia, showing segmental distribution of sensory symptoms.

sensibility to pain, occupied almost the same areas, excepting the back (Figs. 4 and 5). Small areas of anæsthesia on the neck, shoulders, and front of the waist were also seen (see figures). This anæsthesia is not common in the disease, and in this case occupied but a limited area. On the right thigh, instead of analgesia, there was hyperæsthesia. This observation is

perhaps of some significance, in view of the fact that the sensory symptoms peculiar to syringomyelia were more marked on the left side, while the motor symptoms were more marked on the right. These sensory symptoms show the segmental distribution commonly seen in this disease.

FIG. 4.



Syringomyelia, showing segmental distribution of sensory symptoms.

Deviation of the spine was a noticeable feature in this patient. It is a very common symptom of syringomyelia, and, according to Bruhl, exists in at least 50 per cent. of cases. This scoliosis, in my case, affected even the cervical spine and the position of the head, giving the head and neck a twist not unlike that seen in torticollis. This is shown to advantage in the photo-

graph (Fig. 1), which shows also the weakness of the neck muscles, which made it necessary for the patient to have his head supported by an attendant while being photographed. When the patient was lying down this scoliosis was almost obliterated.

Trophic disorders. The right ankle was enlarged, but not so much so as formerly, according to the patient. It was undoubtedly an arthropathy similar to those seen in locomotor ataxis, general paresis, and multiple sclerosis, to which diseases and to syringomyelia true spinal arthropathy appears to be limited. Grating, denudation of bone, stalactites, and effusion were not noted. It is probable that the arthropathy had been of the milder type, recognized by many original observers, in which there is a tendency to recover. This enlarged ankle is seen in the photograph.

The toe-nails were very much deformed; they were enlarged and thickened, had transverse ridges, and were quite brittle. The patient said that several times some of his nails had dropped off.

The man had deeply pigmented maculæ on the legs, which can be distinguished in the photograph. These spots and blemishes were the results of a slowly advancing trophic process in the skin. These changes in the nails and skin have been observed in other cases of syringomyelia. Like most trophic disorders dependent upon central nervous disease, they do not appear to follow any constant law, and their exact pathology is obscure. In Morvan's disease, however, which is now generally recognized as only a special type of syringomyelia, trophic changes, leading to total destruction of the fingers, the painless whitlow, or "panaris analgesique," of French writers, are characteristic.

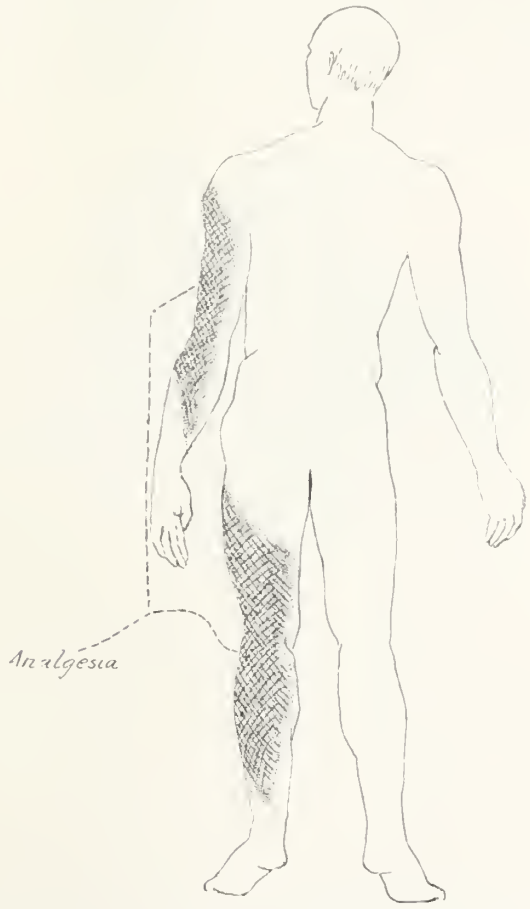
No mental symptoms were observed, nor any symptoms of bulbar involvement. Expansion of the chest was limited to not quite one inch, the left side of the chest being rather freer than the right. I have made a similar observation several times in multiple neuritis, but never before in a case of cord disease. There were no oculo-motor symptoms. The bladder and bowels were innervated normally. The heart was rapid, the sounds normal.

Soon after admission the patient's temperature was noted occasionally to be increased—sometimes to the height of 102°. The lungs were carefully examined, but no evidence of phthisis was found. During August he had an attack of dysentery—which was prevalent at that time in the hospital. This weakened him so much that he was not able afterward to leave his bed even to be propped in a chair. In September I found, on visiting him one day, that he had a severe cough with dyspnœa, which had begun on the preceding day. Examination revealed catarrhal pneumonia, under which he sank rapidly and died the same evening. His embarrassed respiration was doubtless the cause of the rapid progress of the lung complication.

At the autopsy the cord presented the characteristic appearance described by Bruhl. The cervical enlargement was broadened and flattened (somewhat ribbon-like), and to the finger, gently pressing or squeezing it, gave the

impression of containing a cavity. On section, a large cavity was found, beginning in the very lower part of the medulla, broadening out in the cervical region, and extending well down into the dorsal cord. It did not extend into the lumbar enlargement. This canal, or cavity, was very wide

Fig. 5.



Syringomyelia, showing segmental distribution of sensory symptoms.

in the cervical enlargement—so wide in the fresh state that it is not an exaggeration to say that a small penholder could have been inserted into it. In this region it had caused great destruction or alteration of arrangement of the normal constituents of the cord, as seen even by the naked eye. This cavity was not exactly in the middle, but extended rather more to the right

side—a fact which corresponds to the clinical findings. There were no special signs of inflammation or of hemorrhage about the cord.

Sections of the cord have been made from fourteen levels. For this work I am indebted to Dr. Albert A. Ghriskey and Dr. James Homer Wright, of the Laboratory of Hygiene in the University of Pennsylvania.

I wish here to particularly acknowledge my obligation for this work, and to call special attention to these beautiful sections, which have been stained by the Weigert method. The photographs of the sections were made by Dr. W. M. Gray, of the United States Army Medical Museum in Washington, to whom also I am much indebted. The drawings (Figs. 6, 7, and 11) were made by Dr. A. A. Stevens.

DESCRIPTION OF SECTIONS.[1]

General Features.

Under a high power the glioma, stained with methylene-blue, presents numerous neurogliar cells. With Weigert's stain these are not quite so readily seen. They are well seen with a carmine stain. The fibrillary structure, described by some authors, does not appear plainly in these sections. It is apparent in some limited spots, where the tissue is not so dense as usual, but I have not been able to follow fibrillæ from their cells. Nevertheless, the glioma has a mesh-like formation, which shows innumerable minute spaces. Very few vessels are seen in the new formation, and nothing like the single longitudinal vessel of supply described by Bäumler. Most of the vessels are at or near the periphery of the mass, but at least one well-defined one is seen in the cavity. Refractive bodies, described by Bruhl, are plainly visible about and beyond the periphery of the new formation. The cavity has at some points what seems to be a lining membrane, which is even detached in some places, as described by others. This appearance is evidently caused by the shrinking away from the cavity-wall of the solid transparent contents of the cavity, carrying with it some adherent portions of the glia. At some levels a large group of broken down epithelial cells, evidently the remains of the central canal, can be seen. This is situated in front of the cavity and to one side of the centre. It is not connected with the cavity. In the medulla, however, and again in the lumbar enlargement, the central canal, surrounded by gliomatous new formation, can be made out more plainly. There is but little pigment in the cord anywhere. No conspicuous signs of inflammation or hemorrhage can be seen; the membranes are not unduly thickened, nor are the bloodvessels enlarged, increased, nor crowded with leucocytes. Most of the multipolar cells in the anterior horns of the

[1] The author is entirely responsible for these descriptions, to which he has given much attention and care, especially to that of the medulla oblongata, in which the anatomy is complicated and still further confused by disease.

cervical enlargement are atrophied and granular. The lateral pyramidal tracts are densely degenerated, and contain only a few scattered nerve fibres in varying stages of decay. The direct cerebellar tracts to the outside of the crossed pyramidal tracts are much better preserved than the latter. At some levels they contain a sufficient number of normal fibres to make a contrast to the adjacent pyramidal tracts visible in the mounted section to the naked eye. This is significant in view of the connection of these fibres with Clark's vesicular column, and their possible influence on equilibration—

Fig. 6.

Syringomyelia. (Medulla oblongata.)

Ng. Nucleus funiculi gracilis. *Nc* Nucleus funiculi cuniati. *F.* Funiculus cuniatus. *Sub. g.* Substantia gelatinosa. *XI.* Accessory nerve. *DC.* Direct cerebellar tract. *V.* Ascending root of fifth nerve. *Ph.* Posterior horn. *G.* Gowers' tract. *Ah.* Anterior horn. *D.* Decussation of pyramids. *Pyr.* Left pyramid.

The light, unshaded areas are degenerated. The central canal is surrounded with gliomatous material. The fibres of the accessory nerve are too diagrammatic. They are not so distinct in the section.

which was not disturbed in this patient. They are not so well preserved on the right as on the left side; and in the medulla at the level of the decussation of the pyramids, the region of the direct cerebellar tract, as well as of the pyramidal tract before it decussates, is degenerated. The integrity of Clark's vesicular column is difficult to determine, because the region in which it is found is stretched around the end of the cavity in such a way as to distort and confuse the nerve elements.

This cord, followed through its whole length, presents an epitome of the

gliomatous process in all its various stages. Thus, in the medulla the process is diffused in various areas, and a cavity has not yet been formed. In the cervical region the cavity is formed, and is very extensive, with secondary effects in the white matter; in the dorsal region the process is more limited, and the glioma tends to one side; while in the lumbar enlargement the process is still in an early stage, prior to the formation of a cavity.

Section 1 (Fig. 6).—This is the highest section made—at the region of the decussation of the pyramids. The central canal, lined with epithelium, is seen. It is surrounded by gliomatous material, which has not yet begun to break down into a cavity, although under the microscope it is seen to be brittle and friable. The decussating fibres are seen and the remnants of the anterior horns, cut off by them, lying to their outer side. It is to be noted that the fibres running from the right pyramid to the left tract are much more numerous than those running in the opposite direction; also, that the left pyramid is degenerated.[1] The nuclei of the funiculus gracilis and funiculus cuniatus are well preserved. The appearance of cells and ganglion formation can be made out in both. The funiculus cuniatus itself, lying at the head of the column of Burdach, is deeply degenerated on each side. The funiculus gracilis, or head of the column of Goll, is much better preserved. The beginning of the reticular process is visible, although much broken up by glia. The substantia gelatinosa can be seen around the ends of the posterior horns. These posterior horns are of different size and appearance, the left being much the smaller. To either side of the central region (here occupied by glia) is a collection of gray matter with some large cells; and from this region fibres arise and run toward the side of the cord, skirting along the edge of substantia gelatinosa. These are doubtless the fibres of the accessory nerve. In the right lateral region of the cord, occupied by both the direct cerebellar tract and Gowers' tract, the cord is much more degenerated than in the corresponding region on the left. On the opposite side of the cord a small degenerated area is seen also in front of the posterior horn. This is the left direct cerebellar tract, showing here to advantage its triangular shape.

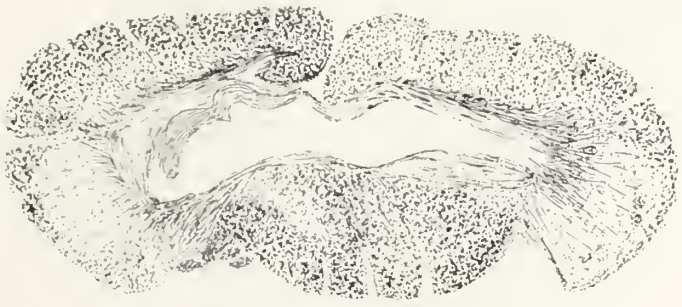
Just below this region a section (not shown in the illustrations) shows extensive gliomatosis, with a very large cavity. The new formation occupies not only the central region, but also portions of the right lateral column and of the posterior columns. The left antero-lateral column is partially preserved. But little gray matter is visible, the cord being a hollow tube with some conducting tracts of white matter preserved.

Section 3 (Fig. 7).—Upper cervical region. Here the cavity extends far across the cord, almost symmetrically on either side. The gliomatous formation extends out along the posterior horns, almost cutting off the posterior

[1] This degeneration of the left pyramid *above* the cavity is worthy of special note. It will be remembered that the patient's motor symptoms were more marked on the right side.

columns from the rest of the cord. The lateral tracts are much degenerated, especially the right and the right direct pyramidal tract. Remnants of the anterior horns can be made out as narrow projections in front of the cavity (the left alone is shown in the drawing); they contain only a few multipolar cells degenerated. The anterior parts, especially, of the posterior columns, are degenerated. But little trace of posterior root-zone is visible. The gliomatous tissue extends almost entirely around the cavity as a rim or circle.

FIG. 7.



Syringomyelia.

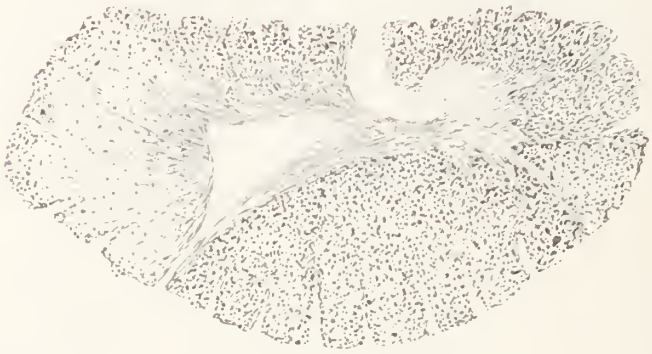
[*Note.*—Many of these general features are characteristic of the segments lower down. The cavity tends to run to the right side in the dorsal cord (as we shall see) and to diminish in size. The lateral tracts will be observed in all the sections to be degenerated to the very end of the cord. The anterior horns are degenerated only in the cervical region. They appear with a full complement of cells in the lumbar enlargement. From the region of the decussation, first described, the central canal is partly obliterated; its epithelial lining, however, is found in almost every section as a group of epithelial cells not connected with the cavity. The cord is distinctly smaller in the cervical region on the right than on the left side. In all, fourteen sections from different levels of the cord were mounted and have been carefully examined. In order, however, to avoid repetition, only a selected number of these are illustrated and described.]

SECTION 4 (Fig. 8). *Photomicrograph.*—The cavity here begins to trend toward the right side (left in the photograph). It is lined with a gliomatous material. Remains of the central canal can be seen in a group of epithelial cells in front of the cavity and to one side of its normal position. It presents irregularly the outlines of a double canal. It does not appear in the photograph. The anterior white commissure is preserved. The gray matter is stretched around the ends of the cavity, and only a few multipolar cells are visible (very indistinct in this photograph) in the anterior horns. The lateral pyramidal tract is deeply degenerated, and the direct pyramidal tract slightly so. The posterior horns and root-zones cannot be well distinguished.

Section 5 (Fig. 9). *Photomicrograph.*—Cervical region next below Section 4. Cavity trends still further to right. (In this the image is not reversed.) The anterior horn can be well distinguished. It contains more multipolar cells than in the former section, but under a high power they are seen to be degenerated. Otherwise the appearances are much the same as in Section 4. The direct pyramidal track on the right is more degenerated than on the left. The anterior portion, especially, of the columns of Goll are involved in gliomatosis.

[*Note.*—In the next two segments, not shown here, the cavity again becomes almost symmetrical, before deviating finally in the dorsal region.]

Fig. 11.



Syringomyelia.

Section 9 (Fig. 10). *Photomicrograph.*—Upper dorsal region. Here the cavity trends to the right (image not reversed). The gliomatous tissue extends down both posterior horns. All around it the white, as well as gray, matter is degenerated, especially in the direct and crossed pyramidal tracts (more marked in the right). The anterior parts of the posterior columns also are affected. The gray matter is almost entirely destroyed, except the anterior horns, which are seen as mere small projections in front of the cavity. They contain but few cells.

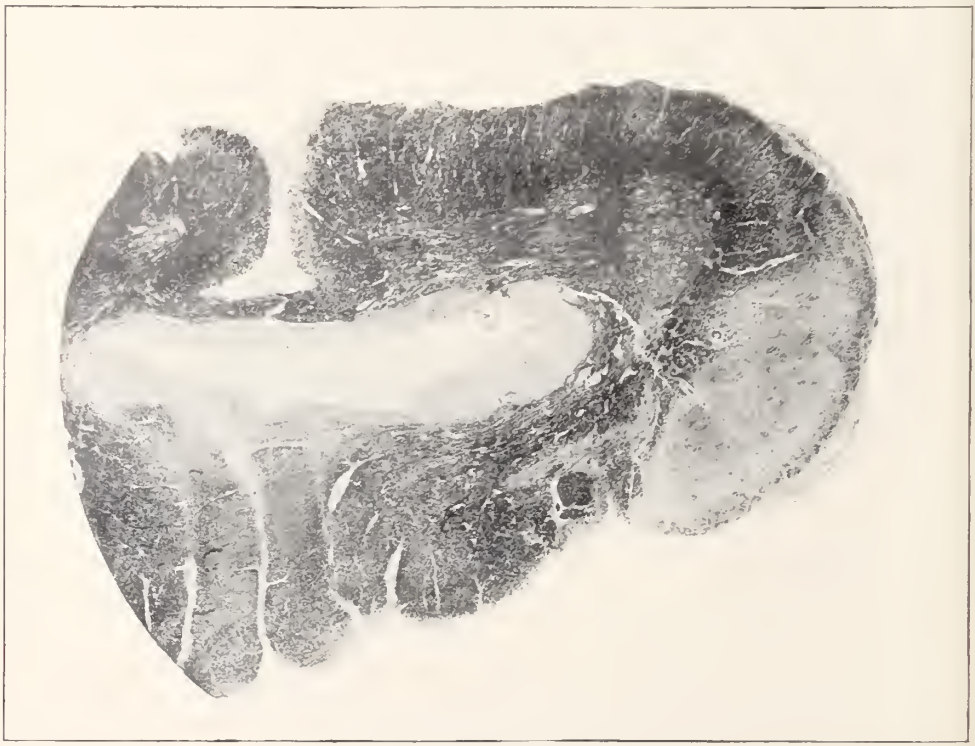
Section 12 (Fig. 11).—This drawing represents very well a section from the mid-dorsal region. (The image is reversed.) The cavity is to the right (left in figure), and follows out the posterior horn quite to the periphery. The right lateral tract is very much degenerated, and the left is rather more so than is shown in the drawing. The right anterior horn is also practically destroyed. The left anterior horn preserves its shape, and under the microscope some motor cells are visible in it. The posterior columns show degenerated fibres scattered through them.

Section 14 (Fig. 12). *Photomicrograph.*—From lumbar enlargement. The cord here presents a striking contrast to its appearance higher up. Its

Fig. 8.

Syringomyelia. Cervical enlargement (section 4).

Fig. 9.

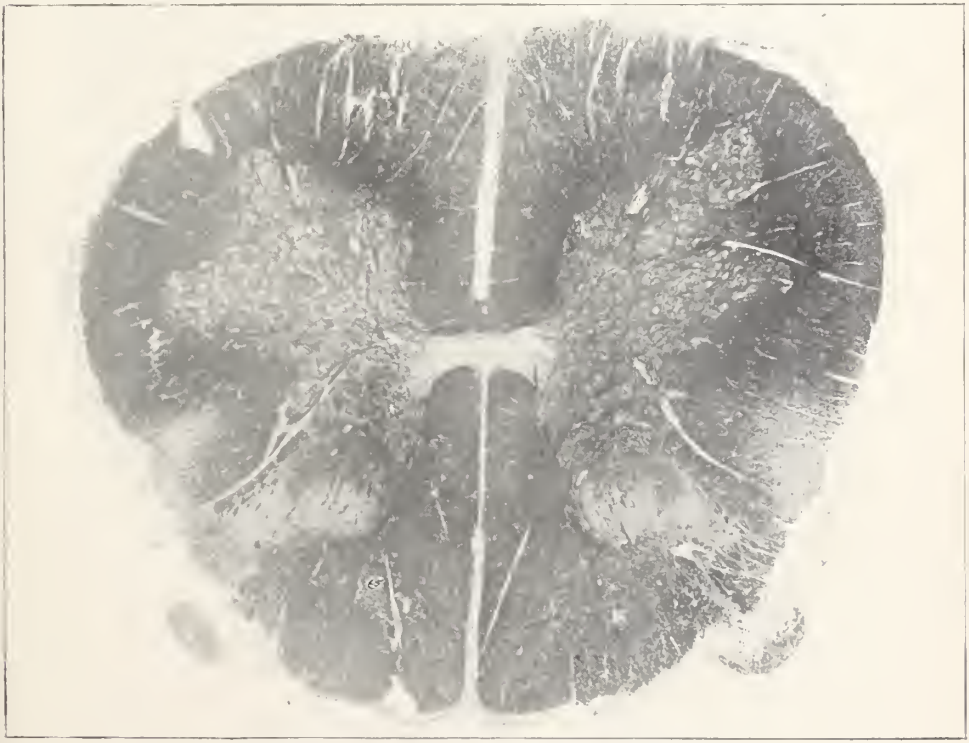


Syringomyelia. Cervical enlargement (section 5).

Fig. 10.

Syringomyelia. Upper dorsal region (section 9).

Fig. 12.



Syringomyelia. Lumbar enlargement (section 14).

general shape and size, as well as those of its gray matter, are normal. The anterior horns are large and well supplied with motor cells. The posterior horns and root-zones are plainly seen. In but two features does it present very marked abnormal appearances; and these, in view of the diseased state of the cord higher up, are full of interest. First, the lateral, or crossed, pyramidal tracts, which are here small and have come to the periphery, are degenerated. Second, the region of the central canal, in the posterior gray commissure, shows distinctly gliomatous change. Already the central canal is obliterated, but no gliomatous cavity has yet taken the place of the normal one. This proves very beautifully that the cavity in syringomyelia is *not* a mere expansion of the normal canal. On the contrary, the normal canal does not exist any longer, and its place is taken by the remains of its columnar epithelial cells, which have undergone coagulation necrosis with loss of their characteristic columnar appearance. This group of broken-down cells is surrounded by the gliomatous mass, which at this level occupies but a small space in the gray commissure. We see here, probably, an early stage of the gliomatous process at the lowest point of its downward progression in the cord. Slight degeneration is seen here in the posterior columns near the periphery.

A few special points occur to me before closing this report.

Already special types and varieties of syringomyelia begin to appear and to be recognized. These are the "formes frustes" of French writers. The most notable of these is the "type Morvan," which, until very recently, was claimed by many to be a distinct disease. Its special symptom is a destructive whitlow, painless and chronic, of the fingers. Joffroy has already proved that this destructive lesion is trophic, and that it depends upon a gliomatous change leading to syringomyelia. Other trophic lesions sometimes dominate the scene, and by appearing early and constituting the most conspicuous features, may be confusing in the diagnosis. Joint changes may thus appear, and be called rheumatic. In my own case it may be recalled that an enlargement of the ankle was the first symptom noted by the patient. This was evidently a true spinal arthropathy. Another lesion is the one noted by Charcot, enlargement of the hand, closely simulating that of acromegalia, which Charcot calls chiromegalia. In his case the change was limited to one hand, and was proved to be symptomatic of syringomyelia.

Other varieties are the asymmetrical cases. These may be

monoplegic or hemiplegic in type. A case has already been reported in which a hemiplegia of spinal origin was the earliest symptom. This doubtless depends upon the cavity extending, as in my own case, more toward one side.

The possible confusion of syringomyelia and some of the cases of so-called Friedreich's ataxia has always been an interesting point to me. In some cases of syringomyelia the posterior root-zones and columns are involved (the secondary extrinsic symptoms already referred to). This fact, together with the existence of scoliosis, and the well-recognized fact that both diseases are probably developmental or embryonal in origin, and the still more significant fact that in three out of the twelve autopsies in cases of Friedreich's ataxia, collected by Griffith, cavities were found—all these facts suggest an analogy and a possibly deep relationship between the two diseases. Déjérine contends that Friedreich's ataxia is due to a gliomatous change.

Probably the most widely received opinion of the exact nature of syringomyelia is that it depends upon the proliferation of an embryonal tissue remaining in that region of the cord in which the medullary folds in the embryo close over to form the central canal. It is thus a developmental defect, and the causes of it reach far back into the intra-uterine life.

That this degeneration is not a mere sclerosis of connective tissue finds a curious confirmation in a work of Chaslin,[1] who observed some special features in the histo-chemical reaction of this gliomatous material which serve to differentiate it.

DISCUSSION.

Dr. Arthur V. Meigs: It may be worth while to call attention to the fact that it sometimes happens that conditions like those described are found post-mortem without any special symptoms having been present during life. I have in my possession a number of sections of the spinal cord of a man who died in the Pennsylvania Hospital from Bright's disease. The post-mortem

[1] Quoted by Bruhl (p. 89).

showed a syringomyelia of considerable size in the cervical region. There had been no symptoms referable to this condition.

There are in the National Museum in Washington a number of photographs of sections from a case of my own, that of a baby three weeks old who died with spina bifida and paralysis. There was extensive dilatation of the central canal and a large-sized syringomyelia or cyst in one of the posterior horns, quite low down. Of course, in this instance, no symptoms could be noticed. In the adult, a man about fifty years of age, however, there had been no symptoms.

Dr. Lloyd: I would only say in reference to the statement made by Dr. Meigs, that several years ago we were not so well acquainted with the symptomatology of syringomyelia as at present. It requires a careful study of the sensory conditions to detect them. I think that if Dr. Meigs's patient were living now and we had an opportunity to examine him, we should find some sensory and motor disturbances. There may be, in these cases, a slight atrophy of the muscles around the shoulder girdle which does not attract attention.

EXHIBITION OF SPECIMENS FROM CASES OF SYRINGOMYELIA.

By WHARTON SINKLER, M.D.

The first specimen is from a patient, aged fifty-five years, who was in the Philadelphia Hospital for several years, and who died in August, 1892, from syringomyelia. He was by occupation a butcher, which is a point of interest, as some have stated that this affection is more liable to occur in persons who use the upper extremities to a large extent. When admitted to the hospital, in July, 1886, it was found that there was wasting in the upper extremities, and with a tendency to contracture of the extensors of the hands and fingers. The loss of power in the flexors and the preservation of the tone of the extensors gave rise to a singular deformity, similar to that of the hands in cervical pachymeningitis. The muscular atrophy increased steadily, and in March, 1892, when I delivered a clinical lecture upon him, there was excessive wasting of the upper extremities, exaggerated knee-jerk, and clonus. Well-marked thermal anæsthesia was present in the arms, and, as shown in the diagram which I present, in areas over the trunk down to the pelvis. The sense of touch and the pain sense were preserved. The patient died from exhausting diarrhœa, and the autopsy showed the characteristic conditions of syringomelia. There is, as you see, flattening of the cord, with a large cavity beginning in the cervical region and extending throughout almost the entire length of the cord. This cavity in the cervical region has absorbed almost all of the gray matter of the cord. There is but little gliomatous tissue around the cavity.

For the opportunity of exhibiting the second specimen I am indebted to Dr. Weir Mitchell. The patient was a man, twenty-seven years of age. Never had syphilis. Six years ago he had a severe attack of pain in the nape of the neck, lasting six weeks, and requiring much morphine. Immediately afterward he had typhoid fever, and has never been well since. During convalescence he had a second attack of pain in the nape of the neck lasting two weeks. The pain was cutting, severe, and continuous, worse at night, and extended across shoulders and down arms to elbows. Has had several similar attacks since. Two years ago had a febrile attack confining him to bed five weeks. A few months later he noticed right arm was weaker than the left arm, and in about a year weakness appeared in right leg. Ap-

petite good. Bowels sluggish. No loss of sphincter control. No headache. Vision and hearing good.

Present state: Well nourished. Right arm is very feeble. Can abduct by deltoid, can flex forearm on arm, can flex and extend wrist, flex and extend fingers a little; cannot do much more than just move thumb toward palm. Pronation and supination fair.

The left arm is one-fourth inch greater in circumference than the right. He is right-handed. There is no marked interdigital wasting; right thenar and hypothenar eminences are wasted. Nails are not ridged. Hair normal. Muscles flabby. Right pectoral smaller than left. Right elbow jerks absent. Muscle irritability plus. Dyn. R. 5, L. 59.

Sensation to touch and pain impaired in right hand. Temperature sense perverted in both hands.

Ankle clonus. Achilles jerk and plantar reflex are present on both sides. Muscular irritability plus on both sides. Cremasterie and abdominal reflex absent. Spinal column straight. No deformity. Quite marked tenderness on pressure over the fourth cervical spine.

Dr. de Schweinitz examined eyes and reported no lesion of consequence.

Upon the supposition that the symptoms were due to a tumor of the cord in the cervical region, the operation of laminectomy was performed by Dr. W. W. Keen. The spinal canal was exposed in the cervical region, but no growth was found which could be removed.

Patient died on the eighth day after operation.

Autopsy: The spinal canal in the cervical region was completely filled by the cord. On removal of the cord it was found to be the seat of a tumor occupying the entire cervical portion of the cord. The cord was here much swollen and distorted in shape. On section it was reddish in color and infiltrated in spots with blood. A cavity, central in position, extended from the fifth cervical down as far as the fourth dorsal nerve, and this cavity was surrounded by a glioma. The cavity became smaller from the cervical cord downward, and from the dorsal region down the central portion of the cord was occupied by the glioma. There was descending lateral degeneration in the dorsal and lumbar cord. There was marked acute myelitis at the seat of operation.

These two specimens exhibit strikingly the two forms of syringomyelia. The first is the form in which there was a congenitally dilated central canal which progressively became larger during life with the development of a gliomatous infiltration surrounding the cavity, while in the second specimen we have a gliomatous tumor which originally occupied the centre of the cord, which has undergone degeneration and absorption in the centre with the formation of a cavity.

A SECOND CASE OF LIVER ABSCESS DUE TO DISEASE OF THE VERMIFORM APPENDIX.

By GEORGE ERETY SHOEMAKER, M.D.

[Read February 1, 1893.]

At a late meeting of the Society the writer presented a specimen showing a pin in the vermiform appendix, which was removed from a young man who had peri-cæcal abscess and also abscess of the liver. It was then suggested that the abscess of the liver was due to infection from the appendix which had travelled along veins belonging to the portal system to the liver, this position being strengthened by a line of infection visible to the naked eye from the root of the mesentery to the transverse fissure of the liver. By one of the coincidences so often observed in practice, the writer is enabled to present a second case, with specimens showing gangrene of the appendix and multiple abscesses of the liver, which are undoubtedly secondary. The cases are interesting because abscesses of the liver due to disease of the appendix are unusual, and of late years appear to have received little notice, at least from American and English writers. The following is almost a typical case of pylephlebitis suppurativa, or suppurative inflammation of the portal vein, as described by Frerichs.[1]

W. S., seventeen years of age, a packer, had been previously healthy. His father died of enteric fever and the mother of apoplexy, though a year before her death I removed a sarcoma of the breast and axilla.

On November 12th, after eating indigestible food for breakfast and dinner,

[1] Clinical Treatise on Diseases of the Liver, 1861.

the patient worked for some hours in an unusual draught of cold air, and was seized with pain just below the navel, together with nausea, followed in the evening by vomiting. Two solid stools passed that day.

On the 13th the abdominal pain became localized in front of the anterior superior spinous process of the ilium on the right side, passing a little backward along the iliac crest. There was some difficulty in fully extending the right thigh. He had a severe chill in the evening, followed by sweating and vomiting. He had one solid stool.

On the 14th localized pain continued. There was some nausea and gulping, but no vomiting; he had two severe chills (shaking his bed distinctly) in the evening, and two in the night, followed by profuse sweating. He had one loose stool.

I saw him for the first time on the 15th, finding him up and dressed at 9 A.M., with a temperature of 99° and a pulse of 90; the conjunctiva was yellowish; there was decided tenderness over the appendix, with resistance of muscles, but no tumor and no pain, even on full breathing; there was no tympany, and his mind was unusually clear. The diagnosis of appendicitis was announced, and the family informed that operation would have to be considered, though not at that moment demanded.

The patient now passed out of my hands, and I am permitted to report his subsequent history by courtesy only. He was admitted to one of the hospitals, where he had immediately a violent chill, followed by a temperature of 104° and sweating. Until this time the diagnosis did not appear at all obscure, but when seen next day by the visiting surgeon, the presence of appendicitis was not deemed positive, because local tenderness had disappeared from the right iliac fossa; nor was it developed by rectal pressure. There was not even thickening to be felt by the rectum, and the resistance of the abdominal muscles was slight. The abdomen was not only flat, but scaphoid.

From this time on until death, eight days later, the obscurity of the diagnosis deepened. Jaundice set in and soon became intense; there was almost complete insomnia, but no delirium. Full doses of quinine seemed to moderate the chills, while small doses of calomel were followed by frequent, thin, greenish stools. There was a very irregular temperature, reaching a little over 103° or dropping to 99°. The liver was somewhat enlarged but not tender; the spleen was enlarged. Rapid emaciation followed, with brown, dry tongue; dulness succeeded the abnormal brightness of intellect, and the patient died a few hours after he had been transferred from the surgical to the medical ward, and just twelve days after the onset of the disease.

Autopsy. Emaciation was deep and there was universal jaundice. The heart was normal; the lungs were moderately adherent at the apices, without abscesses. The kidneys were large, congested, the capsule stripping easily; no pus was present. The spleen was about twice the normal size and contained at least one hemorrhagic infarct as large as a pea. The intes-

tines were not distended. There was no sign of general peritonitis; white gauze dipped in the scanty serum at the bottom of pelvis stained clear yellow. On lifting the cæcum and separating extremely slight adhesions, about two drachms of thin, gray, flocculent pus appeared about the appendix, which was thickened and gangrenous in its middle third, a slough about three-quarters of an inch in length having dropped out of the posterior wall. A concretion, one-quarter of an inch in diameter, afterward shown to be fecal and not a gall-stone, occupied the upper and healthier third of the appendix, completely filling its lumen. An area of parietal peritoneum upon which lay the appendix was grayish-black and gangrenous, showing a small point of perforation. Behind the peritoneum and extending obliquely upward from the root of the appendix toward the front of the spine was an infiltrated area of loose connective tissue and veins, which could be picked up within the abdominal cavity like a section of bowel. This, when cut across, was seen to be inflammatory, the distended veins within it having thickened walls with a dirty-gray lining, and containing a grayish fluid. The liver was somewhat enlarged, and was studded throughout with abscesses from a line to an inch in diameter. These were very numerous about the transverse fissure. The gall-bladder was distended by a mucoid fluid. No gall-stones were found. The brain was normal. No pus was found elsewhere. There were numerous ulcerations of the mucous coat of the cæcum, the majority about two lines, and the largest a third of an inch in diameter. One of these encircled the opening of the appendix into the cæcum. This opening barely admitted a probe.

The points of interest in this case are two: Its obscurity from a diagnostic point of view after the liver-symptoms appeared; and the secondary venous and hepatic involvement.

The clinical history emphasizes the fact, well known to many, that the most serious disease of the appendix, going on to gangrene and perforation, may exceptionally exist without the traditional signs of peritonitis; without tenderness on deep pressure, without a demonstrable tumor, without tympany and without pain, at least in the later stages. There can be no reasonable doubt that operation on the third or fourth day would have saved this man's life by forestalling the infection of the veins, just as it would have killed him, or, at least, would have done no good, after the pyæmic symptoms had set in. It is such cases as these, growing more obscure as the patient grows worse, that add force to the reasoning of those who would operate at once in every case as soon as the diagnosis of appendicitis is made. While there is, however, os

large a preponderance of recoveries from attacks which do not go on to pus-formation, that position seems to me to be extreme.

The diagnosis of suppurative inflammation of the portal vein, or pylephlebitis suppurativa, is a matter on which there is no small difference of opinion. Some writers consider a diagnosis during life to be a mere matter of guesswork, while others consider it quite feasible. As has been suggested by Von Schüppel, this discrepancy can be accounted for by the obscurity or clear character of the cases individual writers may have seen. As few men have seen more than two or three cases, they are likely to generalize from these. The following points are given[1] as the most important in recognizing the condition:

1. "The presence of an affection which we know from experience may act as a starting-point . . . especially perityphlitis, a purulent focus, or an ulceration of the stomach, intestines," etc.

2. "Pain in the epigastrium, above the umbilicus, or in the right hypochondrium, or any other situation in which the pylephlebitis may start."

3. "Violent chills, which are repeated at irregular intervals, and are followed by great heat and profuse sweats, while the temperature in the intermission remains abnormally high (and pyæmia in the ordinary surgical sense is excluded)."

4. "The recent, uniform, and painful enlargement of the liver. Enlargement of the organ is not constant, but tenderness is always present."

5. "Considerable enlargement of the spleen, especially when we can follow its development.

6. "The icteric color of the skin and urine in addition to the biliary diarrhœa."

7. "Rapid emaciation of the body and profound loss of power."

8. "The occasional development of profuse peritonitis and typhoid symptoms in the later stages of the disease."

[1] Ziemssen: Cyclop. of the Practice of Medicine, ix, 822 et seq.

Jaundice is said to occur in three-fourths of the cases by Von Schüppel. Eichhorst[1] says that it is almost always present, while in liver-abscess, not pyæmic, it is one of the rarer symptoms. For example, Rouis[2] found it to be present in 17 per cent. of the 258 tropical cases included in his statistics. Waring[3] found it in somewhat less than 6 per cent. of his cases, which were also tropical.

All observers note the violence of the chills in pylephlebitis. There is seldom time for the development of ascites or of enlargement of the superficial abdominal veins. Only one termination is known for the disease, and that is death. This occurs within two weeks as a rule ; sometimes after five or six.

The abscesses of the liver are usually very numerous and small, from the size of a pin-head to that of a cherry, though they may reach a much larger size, as in the following case :[4]

A French soldier, previously healthy, had pylephlebitis following appendicitis. At the autopsy there were found several abscesses of the liver, one of them large, involving the whole thickness of the left lobe of the liver. The portal vein was filled with pus to its smallest ramifications. There was jaundice, but no tympany and no general peritonitis.

The walls of the portal vein or of any contributing branches which may be involved are thickened, reddish or grayish; the intima is of a dirty gray, swollen, and at times much softened or completely broken down. The contents of the inflamed veins are described as ichorous, putrid, dirty gray or reddish-brown fluid. The connective tissue adjacent is also frequently inflamed and infiltrated.

All abscesses of the liver were supposed by Budd[5] to be secondary to inflammation or ulceration somewhere in the digestive tract, as in dysentery, ulcer of the stomach, or proctitis. This theory, though for a long time received with hesi-

[1] Specielle Pathologie und Therapie, 4th ed., 1890, ii, 452.

[2] Recherches sur les Suppurations endémiques du Foie, etc. Paris, 1860; p. 189.

[3] An Inquiry into the Statistics and Pathology, etc. Ed. John Waring, Resident Surgeon of Travancore, 1854, iii.

[4] Archives de Médecine et de Pharmacie Militaires. Paris, 1891, xviii, 62.

[5] Diseases of the Liver, 1853.

tation (Flint,[1] Frerichs[2]), is gradually gaining ground, and is probably, in the main, correct. At the time of examination the original disease may not be present, or it may not be found, while the embolus, which was the means of transfer, has long before disappeared in the resulting abscess. Many of these cases of ordinary liver-abscess run a prolonged course, as is well known, and often give rise to little disturbance of the system. They may either rupture spontaneously, or be drained by operation, with a resulting cure in a considerable proportion of cases.

Very different, however, is the clinical picture when from the same starting-point—an appendicitis, for example—the portal vein or its contributing branches become involved in a suppurative inflammation, as in the last case narrated. These cases all die (Osler,[3] Eichhorst, Von Schüppel), and die quickly, with symptoms of the most violent character. The abscesses which form in the liver are multiple, and before large areas have time to break down death occurs from septic intoxication. It is not always that the inflammation spreads to the portal vein by continuity along a contributing branch, but a septic embolus may be the means, as in the tropical abscess. In each case the embolus traverses the same vessels. In the tropical abscess there is at some period abundant involvement of intra-hepatic portal radicles. What is the cause of the different result? It seems to me that it must be sought in the different characters of the infective material. There must be different ptomaïnes involved, probably different bacteria, so that in the one case a comparatively mild and circumscribed hepatitis is set up, and in the other an infective inflammation of all points of contact in liver and vein, which rapidly destroys life by a species of pyæmia. It is these cases which are comparatively rare. An examination of late volumes of the *Index Medicus* will disclose from one to two cases per year reported as pylephlebitis, but seldom in Ameri-

[1] Practice of Medicine, p. 604, 1880.

[2] Loc. cit., p. 116.

[3] Principles and Practice of Medicine, 1892, p. 451.

can or English periodicals, the German predominating. For the same period the literature of ordinary hepatic abscess is enormous. References to some typical cases are given below.[1]

DISCUSSION.

Dr. J. P. Crozer Griffith: It might be of interest to call attention to the difference in the symptoms in suppurative pylephlebitis and ordinary pylephlebitis. This is not an ordinary abscess of the liver but one of pylephlebitis. In ordinary pylephlebitis there is no jaundice, but there is ascites and other symptoms of intense portal obstruction. In suppurative pylephlebitis, on the other hand, there is intense jaundice, but no marked degree of the symptoms of postal obstruction.

I notice that Eichhorst refers to suppurative pylephlebitis as a sequel of inflammation or ulceration anywhere in the gastro-intestinal tract, but especially in the region of the cæcum. He also refers to the fact that jaundice is intense and accompanied by stools that are not clay colored and urine that may not contain bile, although intensely discolored; and he raises the question whether the jaundice in this condition is not a hæmatogenous icterus due to the presence of a septic poison throughout the circulation and a consequent decomposition of blood. Nevertheless, most recent writers have, I think, taken the ground that hæmatogenous jaundice is rare, and that the jaundice in septic cases is probably due to cellular swelling which closes the small biliary capillaries. It would be of interest to know what was the condition of the urine and of the stools in the case reported to-night.

[1] Sonnenfeld: Wiener med. Presse, 1885, xxvi, 1259. Colquhoun: Lancet, London, 1887, ii, 606. Aufrecht: Berlin. klin. Wochenschr., 1869, S. 308. Payne: Path. Trans., 1871. Many references to German and French sources are given in Ziemssen's Cyclopædia of Practice of Medicine, ix, 805.

TREATMENT OF CHRONIC VALVULAR DISEASE OF THE HEART.

By JAMES TYSON, M.D.,
PROFESSOR OF CLINICAL MEDICINE IN THE UNIVERSITY OF PENNSYLVANIA.

[Read March 1, 1893.]

Since there are certain points in the treatment of disease of the cardiac valves which are the same for the different orifices, I shall consider first such measures as are thus common, referring more especially to mitral and aortic disease.

In the first place, it is well known that there exist chronic valvular defects at each of these orifices which give rise to no symptoms whatever, and are often accidentally discovered. From the standpoint generally conceded, that such defects themselves are irremediable, it is clear that, in the absence of symptoms, medicinal treatment is quite unnecessary. On the other hand, it is a happy circumstance when the subjects of such lesions are made aware of their presence, because they are enabled so to regulate their mode of life as to prevent harmful consequences, either symptomatically or organically. Such persons should avoid over-exercise and excitement. Running, or even walking rapidly, hurriedly ascending stairs, extremes of passion of all kinds, and especially of anger, should be avoided, as also exposure and irregular living. There is a second grade of involvement of either orifice which demands the same treatment in a more imperative manner, since its omission results in a loss of compensation, manifested by dyspnœa, palpitation, and præcordial distress, promptly relieved by the treatment above specified.

In a more advanced degree of interference with normal function, the treatment becomes different with the seat of the lesion. Let us begin with a consideration of lesions of the mitral valve, and first, the most common of all forms—*mitral regurgitation*. The blood flows back into the left auricle during systole of the ventricle, at a time when all communication between these cavities should be cut off and the movement of the blood should be forward only. Averted for a time by hypertrophy of the left auricle, engorgement of the lungs ultimately results, with defective aeration of blood and consequent shortness of breath. This effect is at first counteracted by the increased effort of the right ventricle, whence its hypertrophy, with sharp accentuation of the pulmonary second sound heard at the left edge of the sternum.

So long as compensation is maintained by the supplemental action of a hypertrophied left auricle there is probably no sign of embarrased breathing, or irregularity, or præcordial oppression, or digestive derangement; but as soon as the auricle begins to fail the engorgement of the lungs begins, and the hypertrophy of the right ventricle comes to the rescue. And even *it* may, for a time, by the reinforcement it gives to the left auricle, effect the required compensation. But a suspension of the conditions which co-operate to help it, or a slight yielding of its muscle to the resistance to be overcome, demands assistance. The heart-tonics, of which digitalis is the type, are the agents pre-eminent for this purpose. That they operate by directly increasing the force of the right ventricle and left auricle, and thus contribute to the compensation, can scarcely be doubted; but that they help also to make the closure of the mitral orifice more complete, by forcibly increasing the contraction of the left ventricle, seems also reasonably sure, since the experiments of Ludwig and Hesse have made it so plain how this can occur. They have shown that the mechanism for closing the mitral orifice does not reside in the valve alone, but that the surrounding muscles of the ventricle have an active share, not only in floating up the valve curtains, but also in reducing the size of the opening which

these valve curtains have to close. This is, of course, less applicable in chronic valvular conditions where there is stiffness from calcareous change than where regurgitation results from simple feebleness of muscle in anæmia and after the infectious fevers.

The effect required of this class of drugs varies with the degree of obstruction to be overcome, and the doses vary accordingly. Very often the heart requires but little steadying to enable it to accomplish the desired end, and moderate doses —such as five minims of the tincture of digitalis once in eight hours—suffice. On the other hand, it is a mistake to give too small a dose, and too great timidity often results in failure. Doses of fifteen minims of the tincture of digitalis, every three hours, and corresponding doses of the other preparations are often necessary, and sometimes produce magical effects. The irregular and halting pulse becomes regular, the dropped beat is again taken up, the dusky lips become pink, the scanty urine is increased, the shortness of breath disappears, and calmness and quiet succeed distress and restlessness. As soon, however, as the desired effect is produced the dose should be lowered. The same principles apply to the management of the still more serious engorgements of the venous system which succeed upon tricuspid insufficiency, and produce dropsies and serous effusions. It is in the effort to drive the blood through the engorged veins that the left ventricle finally hypertrophies in this form of disease.[1] Its failure to accomplish this through its inherent power demands the same remedies which the right

[1] At this point I may allude parenthetically to the matter of hypertrophy of the left ventricle in mitral regurgitation. It has always been somewhat a matter of conjecture as to precisely when it occurs. It is commonly said that it is the result of the hypertrophy of the left auricle reinforced by hypertrophy of the right ventricle. But while these agencies doubtless contribute to a distention of the left ventricle and tend to dilatation, it is doubtful, to my mind, whether they tend to hypertrophy. For the idea of hypertrophy implies increased resistance, and there is no increased resistance with a patulous aortic orifice and a patulous mitral orifice, and it is only when the movement of the arterial current becomes delayed by the full veins that this condition of increased power, and therefore of hypertrophy, comes into play. So that it appears to me that while distention and enlargement of the ventricle is an early result of mitral regurgitation, hypertrophy is not.

ventricle needed at an earlier stage to overcome the lung engorgement, and here it is that the large doses of cardiac stimulants are demanded.

This engorgement is also relieved by the use of purgatives, and as the portal area, including the liver itself and the stomach, is especially involved, mercurial purgatives are especially indicated. Five to ten grains of blue-mass at bedtime, followed by a saline in the morning, relieve the congestion, and with it the nausea and indisposition to take food which attend it. Such remedies may be resorted to semi-occasionally. Sometimes the continuous use of small doses for a long time —say one-half to one grain of blue-mass three times a day— is more efficient. The old-fashioned combination of calomel, squills, and digitalis—in doses of one-half grain of the first and one grain of the second and third, three times a day, or smaller doses more frequently repeated—is sometimes most happy in its effect. Digitalis is a remedy always better intermitted to obtain its best effects, and a remedy, too, which, having once excited nausea, is thereafter badly borne. It does, however, sometimes happen that digitalis may be given continuously in moderate doses—say five minims three times a day—with great advantage, while its omission is followed by signs of failing compensation. It is generally recognized that digitalis produces also contraction of the arterioles, and that through this, in connection with the forcible systole, the arterial pressure is increased. This effect is desirable and useful in the early stages of mitral regurgitation, before tricuspid regurgitation and dropsy have set in. Later in the disease, however, when dropsy has set in, this effect militates against the diuretic action which is so much needed. How this may be overcome will be mentioned later.

Before leaving the subject of the heart-tonics in mitral regurgitation, it may be worth while to spend a few minutes on that of the relative value of the different preparations of digitalis. While testimony is generally favorable to the infusion as the most efficient remedy, yet, on account of convenience and accessibility, the tincture is mostly used. I have on many

occasions reiterated that I was inclined to believe that the greater apparent efficiency of the infusion was partly due to the fact that it was generally given in larger doses. Thus, a tablespoonful, or half ounce, is not an infrequent dose of the infusion, while 10 minims, or 20 drops, of the tincture and 1 grain of the powder are not often exceeded. When it is remembered that a half-ounce of the infusion, as made by the U. S. Pharmacopœia, represents nearly 3 grains of the powder, or 20 minims of the tincture, one may understand why it is more efficient. Recently, however, I have thought the infusion better borne by the stomach than equivalent doses of the tincture. It may be that the cinnamon-water with which it is made has this effect.

Of remedies which may be substituted for digitalis, strophanthus should, perhaps, be first mentioned; not that it is always the best. Great expectations were excited when the results of strophanthus were first published by Fraser. It will be remembered it was reported as having all the effects of digitalis on the left ventricle without the contracting effect on the arterioles. The expectations entertained were not, however, realized by clinicians, and it soon fell into partial disuse. Recently I have resumed the use of strophanthus in much larger doses, having given as much as 10 minims, or 20 drops, every two hours for forty-eight hours, without interruption, and with good results. It is undoubtedly better borne by the stomach than digitalis.

Caffeine is an admirable heart-tonic in mitral regurgitation. I do not give less than 3 grains at a dose of the citrate, but seldom give more, every three hours. When caffeine has been given in full doses for some time it produces mental symptoms quite characteristic, consisting in hallucinations not unlike those of delirium tremens, the patient imagining there are persons, animals, and other objects about him, and he is sometimes difficult to control. They, however, cease immediately when the drug is discontinued. Another effect of caffeine, which sometimes interferes with its usefulness, is its effect in inducing insomnia.

Sparteine sulphate is another heart-tonic which I have come

to value very highly, especially where a diuretic effect is desired. The dose I have come to rely upon, after a good deal of experience, is never less than $\frac{1}{4}$ grain, increased to $\frac{1}{2}$ grain, three, four, and five times a day.

In the much rarer disease of simple *mitral stenosis*, compensation is even easier and longer maintained by nature's own resources than in mitral regurgitation. Here, for evident reasons, there is no tendency to dilatation or hypertrophy of the left ventricle. On the other hand, hypertrophy of the left auricle becomes a conspicuous condition, succeeded by hypertrophy of the right ventricle, for the same reason as in mitral regurgitation.

Especially easy is it to maintain compensation if the narrowing is not too great, and if there is a well-preserved left auricle and a strong right ventricle. If, however, the mitral narrowing is extreme, it is plain that the pulmonary engorgement will become greater if we increase the force of the right ventricle. Much more cautious must we be, therefore, in the use of digitalis. Much more needed under these circumstances is relief to the pulmonary congestion, which in turn will relieve the right heart-tension. For the same purpose aconite is sometimes of advantage in these cases in the shape of small doses, say 1 minim or $1\frac{1}{2}$ minims every two hours or every hour, watching its effect. It is possible that it is through a somewhat similar action that convallaria majalis—a remedy in which most observers have been disappointed—has been found useful by Dr. Sansom[1] in mitral stenosis, and also by the French school, as represented by Germain Sée. By these observers it has been found diuretic, increasing the twenty-four hours' urine to 85 and even 115 ounces, reducing the pulse-rate, regulating irregularity, and improving the breathing, even when accompanied by tricuspid regurgitation. The doses given are 10 to 20 minims of the tincture three times a day, and it may with advantage be associated with caffeine, which alone sometimes acts better than digitalis. More effectual than either of these remedies to relieve pulmonary congestion is blood-letting,

[1] "Treatment of Some of the Forms of Valvular Disease of the Heart" (Lettsomian Lectures, 2d ed., with corrections, London, 1886).

and repeated small bleedings are often of great advantage in this form of chronic valvular disease.

The principles governing the treament of combined mitral regurgitation and stenosis are rather those of mitral regurgitation than of mitral stenosis.

And what shall be the treatment of *pure aortic disease?* It will be remembered that both aortic obstruction and regurgitation give rise to hypertrophy of the left ventricle, and that this is compensatory in character, for a time quite sufficient to ward off any unpleasant symptoms, and for a still longer time competent to do this when associated with a quiet life, the absence of excitement, of exposure, and of privation. Its well-marked degrees are accompanied with a powerful systolic impulse, a symptom which is of itself at times a source of great discomfort. Shall we, then, give heart-tonics which increase the force of this thumping blow? Certainly not. Shall we give aconite or veratrum viride, which slow the heart and diminish the force of its stroke? Not as a rule. But there are times when these remedies are indicated. When, as the result of over-exertion, or undue excitement, or gastric derangement, the heart is turbulently overactive, and even irregular in its rhythm, then often I have seen aconite in small doses—say one minim, or two drops, repeated every half-hour or so under close observation—act happily, especially when combined with bromide of potassium, say fifteen grains. The tincture of veratrum viride may be given in slightly larger doses. As soon, however, as this period is past, the aconite should be omitted.

We want rather in this condition to find remedies which will tend to maintain the integrity of the heart-muscle. Such are strychnine, iron in small doses, arsenic, and nutritious, easily assimilable food. Especially useful are well-ventilated living- and sleeping-rooms, wholesome out-door life, with moderate, deliberate muscular exercise. On the other hand, the mountain-climbing advocated by Oertel seems irrational and dangerous. Everyone who has had experience knows that the high altitudes are not well borne by cardiac cases of any kind.

Such measures as these tend to ward off the next stage, for sooner or later the integrity of the muscle of the ventricle yields, dilatation is added to hypertrophy, the auricular ventricular orifice enlarges, and we have mitral regurgitation. Then the treatment becomes that for mitral disease.

The treatment of aortic regurgitation and of aortic stenosis with regurgitation is similar to that of aortic stenosis.

Treatment of dyspnœa. As the dyspnœa is primarily the result of deficient blood-aeration in the congested lungs, the same remedies which force the blood through these organs, and thus relieve the congestion, tend also to relieve the dyspnœa, and often do so. When the dyspnœa persists, it is frequently caused by effusions into the pleural cavity, which are most promptly and successfully removed by tapping, although a blister may also answer the purpose. Repeated tapping may be necessary. Dyspnœa not thus relieved demands an opiate, and of opiates, under these circumstances, morphine is the best. One-quarter grain at bedtime, by the mouth or hypodermatically, gives unspeakable comfort. Hoffman's anodyne will sometimes relieve the milder degrees, and should perhaps be tried first, as it is always desirable to put off the use of morphine as long as possible. Sulphonal may be tried in full doses of fifteen grains. My attention has recently been called to the usefulness of oxygen inhalations in the dyspnœa of heart disease, but I have as yet had no opportunity to try them.

Treatment of dropsy. In like manner the measures that relieve the congestion and dyspnœa tend also to relieve the dropsy, but special means are also necessary. Here it is that full doses of digitalis are especially indicated, and at close intervals, every three hours, and even every two hours. But these measures are often insufficient. And I have become satisfied, from actual and repeated experience, that an essential condition of the successful treatment of the more obdurate of the cases is the restricted ingestion of liquids. With the tissues water-logged and secretion insufficient, it is plain that copious liquid ingestion only increases the difficulty. The principle of the Matthew

Hay method is correct, but in practice it is impossible, because, with an already congested stomach, solids cannot be digested without an admixture of liquid, and further embarrassment results from the effort to dissolve them and from the presence of undigested residue. Therefore, I not only omit solid food altogether, but reduce the liquid to a minimum that will sustain life—not more than two ounces every two hours, and that only during the waking hours. To this I add the use of purgatives. While diuretics sometimes fail us, we can always secure an effect from purgatives. A daily morning dose of Epsom salts or Rochelle salts is given. Then, when action of the bowels begins, full doses of digitalis, caffeine, or sparteine, associated with nitro-glycerin, are almost sure to be followed by copious diuresis, and when these cases start up it is astonishing what quantities of urine are passed. I attach much importance to the association of nitro-glycerin with digitalis at this stage: $\frac{1}{100}$ to $\frac{1}{50}$ of a grain may be given as often as the digitalis and simultaneously. Elimination by the bowels and kidneys being simultaneously stimulated, the sucking up of the interstitial fluid is greatly favored and often rapidly brought about. If these measures be associated with chest-tapping, which may be required, the diuresis set up is often enormous, while the swelling rapidly declines. As diuresis is established, or hunger sets in, the quantity of milk allowed may be increased, and when the dropsy has entirely disappeared, a cautious return to solid food may be permitted. Diuretics or sodio-theobromine salicylate should be tried in doses of 15 grains in solution, 75 to 100 grains being given in the twenty-four hours.

For irregularity of the heart-action and palpitation, which are more common in mitral disease, belladonna is also a useful remedy. I have had little experience with its combination with digitalis, recommended by Da Costa, but I do know that a belladona plaster placed over the palpitating heart is one of the most efficient agents in subduing it. Nitro-glycerin is often very useful to the same end: $\frac{1}{100}$ of a grain,

rapidly increased to $\frac{1}{50}$ of a grain, three times a day, is the proper dose. Cardiac pain is also sometimes relieved by the same remedy.

DISCUSSION.

Dr. H. A Hare: Several points which have been referred to by Dr. Tyson strike me as being of very considerable interest.

First, in regard to strophanthus. I have come to regard it even more favorably than he does. The only disadvantage which I have noted is its tendency to produce diarrhœa when given in full doses. This disagreeable symptom generally comes on four or five days after the drug is first given, and may be quite persistent. I have found that in the cardiac diseases of childhood strophanthus generally has given better results than has digitalis; whereas the reverse holds true in adults.

In regard to "caffeine craziness," so called, I have never given the drug for a sufficient length of time to develop this condition, because I generally had to stop its administration owing to the great wakefulness that it produced when given in full doses.

I am much interested, too, in what Dr. Tyson has said about sparteine, particularly in regard to the dose. Some clinicians have claimed that the proper dose of this substance was $\frac{1}{20}$ of a grain, while others have given as much as 3 or 4 grains. I do not know how this discrepancy in dosage could have arisen, except through the employment of the impure drug, or some confusion as to the drug itself.

Perhaps some clinicians have used sparteine, which is a liquid alkaloid, therefore readily capable of dilution; while others have used sulphate of sparteine, which is a crystalline substance. I believe myself that the proper dose of sparteine is about $\frac{1}{4}$ of a grain, and that it should be given quite frequently.

In aortic disease I am sure that in many cases I have seen aconite do more good than digitalis. I recall one instance, that of a young man nineteen years of age, who came to the wards of the Jefferson Hospital with all the evidences of severe aortic disease and, as I thought, of failure of the heart. He had been a deck hand on a coast sailing vessel, and had worked hard, and I thought that he had ruptured his compensation. Careful inquiry showed that he had not been doing anything for a month. He was put on digitalis, but continuously got worse, and finally was put to bed and kept there, and no drugs given for a few days. It then occurred to me to try the effect of aconite, and under two-drop doses three times a day an extraordinary improvement took place. I have never seen a case of cardiac disease improve so much under rest and the administration of any drug.

Another point of interest is whether morphine is of more value in mitral or aortic disease. A certain number of clinicians have claimed that it is of more use in mitral disease. I have, on the contrary, gotten as much result in aortic regurgitation where digitalis seemed to make the patient worse, as in mitral disease.

In regard to belladonna plaster. I agree that a belladonna plaster placed over the præcordia produces an extraordinary effect, but I cannot explain it. There are a few belladonna plasters which, in a susceptible individual, will produce some signs of the physiological action of the drug, but we should get as good an effect from the application of the plaster over any other portion of the body as over the præcordia. I see no explanation of this unless it be that the plaster may produce a counter-irritant effect, and reflexly influence the heart. This idea that local applications of remedies influence deep-seated parts needs careful study. It is asserted by gynecologists that ergotin injected into the abdominal wall has more effect than if injected into other parts of the body.

Dr. Tyson: I should have said that insomnia was one of the difficulties encountered in the administration of caffeine.

I feel that the results that I have obtained with spartein have been gotten with what are commonly considered large doses. The dose put down in the text-books is one-twelfth to one-eighth of a grain. A number of years ago I began giving doses of half a grain, repeated until at least two grains were taken in the day, and it is since then that the good effects have been obtained.

PELIOSIS RHEUMATICA IN A BLEEDER.

By THOMAS D. DUNN, M.D.,
PHYSICIAN TO THE WEST CHESTER HOSPITAL, WEST CHESTER, PA.

[Read March 1, 1893.]

THROUGH the kindness of Dr. Okie, of Berwyn, I present to the College the report of this interesting case of peliosis rheumatica. Dr. Okie has given me the following notes made of the case prior to consultation with me:

"I first saw Robert L., aged eight and a half years, January 5, 1893. I found slight congestion at base of both lungs. Carbonate of ammonium, counter-irritation, and cotton packing were prescribed.

"He was much better next day. I then noticed discoloration at top of right ear. As the weather was intensely cold, I supposed it had been frost-bitten.

"*January* 7. Ear much worse, discolored and enlarged, with many spots appearing on face, neck, and thighs. Pain in knees and ankles. Was then told for the first time that the boy had complained of pain in knees several days before he was taken sick. Put patient on salicylic acid and soda, two and a half grains each, every two hours, and wrapped joints in cotton; also gave tincture of chloride of iron, twenty drops, with acetate of potassium, two grains, every four hours. Temperature, 102⅖°; pulse, 120."

Condition, *January* 8, when first seen by me.: Pain in joints improved, but joints still tender, and motion painful. Temperature, 102°; pulse, 120, and feeble. Purpuric spots present on both ears, cheeks, neck, shoulders, back, legs, and ankles, and some spots over trunk; also spots seen on gums, roof of mouth, and throat, which was sore. Swallowing painful, and nourishment taken with difficulty.

Ears, lips, eyelids, and chin greatly swollen and tender. Conjunctiva ecchymotic and œdematous. Some vesicles formed on right ear and cheek, but no tendency to exudative erythema or urticaria. Slight bronchial cough, but no consolidation.

January 11. Slight increase of pain in joints, which quickly yielded on return to salicylic acid. The pain preceded the appearance of a few fresh purpuric spots. Temperature, 100°; pulse, 108. On account of weakness he was given, by suppository, quinine, digitalis, and small doses of opium.

13*th*. Swelling of ear, chin, and eyelids diminished, and general condition much improved.

17*th*. Swelling of joints and tumefaction of face had entirely subsided. Temperature normal; pulse, 100, and feeble. Liquid nourishment taken freely. No diarrhœa or mucous hemorrhages. Anæmia marked. Examination of blood showed 3,000,000 corpuscles to the cubic millimetre, in proportion of 1 white to 500 red; hæmoglobin, 60 per cent.; numerous microcytes, 1½ to 3 micro-millimetres in diameter.

Under continuance of iron, general tonics, and a nutritious diet, the patient rapidly improved; and, February 1st, notwithstanding marked anæmia, was able to walk about the house.

The subject of this interesting clinical picture is the sixth of a family of seven boys; they, as well as the father, suffer more or less from rheumatism in joints and muscles, but have no articular deformity. No tendency to hemorrhage has shown itself except in this little patient and an older brother, aged fifteen years. These have had frequent attacks of epistaxis from infancy and severe hemorrhages after trivial injuries.

In 1888 I opened a large axillary axis for Robert, which bled for some time. A profuse bleeding, lasting several days, followed the extraction of a tooth.

An operation, which I performed on the father in 1890, for necrosis of sternum, was not attended by unusual hemorrhage. Neither father nor grandfather of the patient was a bleeder. The grandfather, however, came from a very remarkable bleeder family, which I reported in the *American Journal of the Medical Sciences*, January, 1883. Of his three brothers, Mordecai died of hemorrhage of the stomach at forty; George, from same cause at twenty; and Davis, from hemorrhage following venesection at twelve.

Prof. Osler, in his *Practice of Medicine*, p. 318, states that "Schönlein's peliosis is thought by most writers to be of rheumatic origin; and certainly many of the cases have the characteristics of ordinary rheumatic fever *plus* purpura. By many, however, it is regarded as a special affection, of which

the arthritis is a manifestation analogous to that which occurs in hæmophilia." This case certainly supports the latter view, as does the fact that the disease sometimes appears in several members of the same family.

DISCUSSION.

Dr. William Osler: With reference to peliosis, I saw a case a short time ago—that of a German who was referred to me by Professor Baumler, of Freiburg. It was interesting from the fact that, although there was no history of bleeding in the family, the man was a bleeder. He was thirty-two years of age. The first bleeding occurred in October, 1881. This was from the nose, and the nostril had to be plugged. He bled from Monday until Friday, and was in bed two weeks. Two weeks after that he had another severe attack, the blood coming from the gum, and then for the first time he had urticaria-like spots on the skin, which became hemorrhagic. In December, 1890, he had an attack with severe pain in the back, but no pain in the joints, but profuse bleeding from the gums. At that time he saw Dr. Jacobi in New York. A short time before I saw him, in February, 1892, he had had another attack of bleeding from the gums and also from the nose. During this time he had a fresh development of the cutaneous hemorrhages, such as are so characteristic in peliosis rheumatica, and which are so well shown in Dr. Dunn's photographs. When I saw the patient the stains were still present on the arms and legs, and he was extremely exsanguine. I have heard from him in the past month: he has remained well, and has been taking Fowler's solution and occasionally iron. The case is interesting from the occurrence of purpuric urticaria in a man who had bled so profusely and frequently.

Dr. Dunn: I wish to state that the subject of peliosis rheumatica is a relative of a bleeder reported by Dr. Reynolds Coates, of Philadelphia, in 1828, in the *North American Medical and Surgical Journal.*

A CASE OF LEUKÆMIA WITH RARE LYMPHOID GROWTHS OF ORBITS AND PAROTID GLANDS.

By THOMAS D. DUNN, M.D.,
PHYSICIAN TO THE WEST CHESTER HOSPITAL, WEST CHESTER, PA.

[Read March 1, 1893.]

H. G., aged eight years, came under my observation in the spring of 1891, in consultation with Dr. Pyle, of Glen Mills, with a stubborn case of granular conjunctivitis. Although slightly anæmic at the time, he seemed to enjoy good health. There was no history of cancer, syphilis, or tuberculosis in the family of either parent. A younger sister made a complete recovery from a severe attack of scarlatinous nephritis a year before. The subject of this report had not been a robust child, but never had any serious illness. He was unusually intelligent and studious. The lids improved under yellow oxide ointment and occasional applications of nitrate of silver solution. For the anæmia, tincture of the chloride of iron and cod-liver oil were prescribed. In autumn he returned to school and studied without discomfort. General condition good.

The patient was again brought to me December 15, 1891, for a slight swelling in the left parotid gland—about one inch in each diameter. This swelling was quite hard, with well-defined outline, and immovable. It impressed me as a growth of unusual position and character. It was not tender, but was slightly adherent to the skin, which was not discolored, except by enlarged subcutaneous veins. The parents noticed the swelling three weeks previous to my examination, but attributed it to a cold contracted at school, where he sat by a broken pane of glass—his left side being exposed. The cervical lymphatic glands were not enlarged. Skin and mucous membranes pallid. A highly nutritious diet, the syrup of iodide of iron internally, and locally the tincture of iodine, were ordered.

Dr. Pyle was again called to the case, January 23, 1892, and found slight increase in the parotid swelling, and an appreciable enlargement of the cervical lymphatic glands of the left side. During this period, the anæmia and loss of flesh steadily progressing in spite of the most carefully regulated diet

Dr. Pyle directed as much easily assimilated nourishment as possible, and the continued use of tincture of chloride of iron, Fowler's solution bichloride of mercury, and, at times, quinine and cod-liver oil. Soon the axillary and inguinal lymphatic glands began to enlarge.

About the middle of March a hardness began to develop over the eyes, with slight thickening in both temporal regions. The pulse was weak, and ranged from 96 to 116. Temperature was never below 99° or above 101° during February and March. He kept about the house, and was frequently on the piazza for play. The anæmia and emaciation steadily progressed.

On April 5 the patient contracted a heavy cold, causing a temperature of 103° and severe sore-throat. I was called in consultation April 12, and found an ashy gray membrane over both tonsils, extending to uvula and soft palate; submaxillary lymphatic glands enlarged and tender; temperature 102°; pulse 130; swallowing difficult and painful; marked emaciation; lips, tongue, and nails blue and very anæmic; occipital, inguinal, axillary and cervical lymphatic glands moderately enlarged, but no tenderness over them. The patient had suffered from attacks of pain in splenic region for some time. The left parotid gland was greatly enlarged, of stony hardness, outline well defined. There were also crescentic bodies over either eye, not firmly connected with lids. These swellings began at nasal end of orbit and extended outward beyond external canthus, the larger end toward nasal angle. These were also of marked hardness but somewhat elastic, free from tenderness, slightly attached to lids, and firmly attached to orbital arches. They caused considerable exophthalmos, but only partially interfered with motion of lids. No conjunctivitis; cornea healthy; vision normal. Similar but flat hyperplasias were found in each temporal region; the outline of these was not so well defined. A careful search revealed no other such growths. Marked deafness was attributed to the closure of external auditory canals by the parotid hypertrophies. Treatment was directed to the recent diphtheritic complication. April 14, the condition of the throat was improved; temperature 101°; pulse rapid, feeble, compressible; submaxillary lymphatics decreased and less tender to touch. Nourishment and stimulants were given freely and well retained. A drop of blood under the microscope showed a decided increase in the white corpuscles. The blood was of a very pale color, resembling sero-pus mixed with blood. This examination confirmed the diagnosis of leukæmia which had been suspected at previous visits by both Dr. Pyle and myself.

On April 17, general condition slightly improved, owing to subsidence of the acute diphtheritic attack. Temperature 100°; pulse 124. At this visit a careful blood-count was made. The result showed one colorless corpuscle to fifteen red; 1,830,000 corpuscles (of which 114,395 were white) to a cubic millimetre, and about 30 per cent. of hæmoglobin.

Both hæmoglobin and number of corpuscles were less than one-third normal amount.

On May 7, I saw him for the last time. The anæmia and emaciation had increased; temperature $100\frac{3}{5}°$; pulse 124 and very feeble. The deafness had also increased, but vision was still normal. Unfortunately, no ophthalmoscopic examination was made. The leukæmic growths had all increased, and a small growth, about one-half inch in diameter had developed in the right parotid gland, directly in front of the ear. The little sufferer had several attacks of epistaxis, and on May 5 had two hemorrhages of the bowels. He grew steadily weaker, dying of exhaustion on May 8. Unfortunately, we were unable to secure an autopsy, or even to obtain a portion of the described growths for microscopic examination. There can be no doubt, however, as to the diagnosis of leukæmia.

After a careful search I have been able to find the report of but few cases of similar leukæmic growths.

Doubtless there are others, but the scanty literature of the subject is indicated by the absence of any reference to this condition by modern writers in our leading medical works.

Dr. Henry D. Noyes (*Diseases of the Eye*, p. 666), however, states a case of lymphoid growths of both orbits in Hodgkin's disease. This case was reported in full in the New York *Medical Record*, by Dr. Richard H. Derby.

Cases have been carefully reported by Chauvel, Leber Osterwald, and Fröhlich. The fact that I have been unable to find any reference to these in English, together with their very striking similarity to the clinical features herein presented, justifies a somewhat full abstract of them.

Chauvel's case (reported in *Gazette hebd.*, No. 23, 1877). Patient aged forty-one years, custom-house officer, was admitted to Military Hospital at Val de Grâce, October, 1876, with a tumor of left side of face. Previous to this his health had been good. No history of syphilis. In July, 1876, a small swelling appeared in left upper eyelid. It developed rapidly, and was free from tenderness and pain. By September it became painful. On admission to the hospital the swelling had extended to the cervical lymphatic glands of both sides, which formed a chain the length of the sterno-cleido-mastoid. Some tenderness and redness existed at most prominent points above the left eye. Ulcers were found on left buccal surfaces. Neuralgic pains of left side of face and head existed, with profound cachexia. Tonic treatment and sedative applications were resorted to without benefit. Diagnosis by Professor Perrin, sarcoma of face with symptomatic adenitis.

On November 12, he came under the care of Chauvel. The tumor occupied left supra-orbital region and left upper eyelid, extending from slightly above

eyebrow to root of nose. Left jaw also swollen. Left eye completely closed. The tumor was elastic and hard, and seemed firmly attached to subjacent parts. There was slight deafness. Patient saw black spots before right eye. Ophthalmoscopic examination revealed normal media. Papilla also normal, but outside of disk were several hemorrhagic spots, one large one above and one below. Professor Perrin states that the appearance of the retina was that of hemorrhagic retinitis, the result of the general state and, possibly, of leukæmia. November 19, had slight hemorrhage of the bowels. Died November 21, from exhaustion.

The tumor of upper eyelid was composed of cellular elements, analogous to those of lymphatic glands; these were united without apparent stroma. The growth extended to all parts of the eyelid and was not encysted. Teasing the tissue disengaged reticulum similar to that of lymphatics.

Unfortunately, the medulla of the bones was not examined. The only lymphatic glands involved were those of the cervical region. The spleen was somewhat enlarged, liver greatly so; cells not diseased; interlobular capillaries were dilated and filled with leucocytes; masses of leucocytes were also found in interlobular spaces—changes which characterized the leukæmic liver.

The right side of heart was filled with chicken-fat clot. Large quantities of leucocytes were found in the vessels of the heart, but the organ was free from disease. Unfortunately, no microscopical examination of the blood was made, but the marked anæmia, enlargement of lymphatic glands of the neck, and the accumulations in the vessels of the heart and hepatic glands make the diagnosis of leukæmia indisputable.

Leber's case (*Archiv. Ophth.* No. 24, 1877, p. 295). C. F., aged forty-eight years, with the exception of an attack of rheumatism, had enjoyed good health prior to August, 1876. No history or evidence of syphilis or malaria. At that time he noticed a swelling of under lids, and later, of upper lids of both eyes; he also suffered from languor and weakness. In September was treated by an oculist with iodine, internally and locally without benefit. Had an abnormal prominence of eyeballs. On account of eye trouble appeared before Göttingen eye clinic, April, 1877. His condition was striking and unusual. Lids of both eyes enormously enlarged in all diameters, on account of elastic growths which were not adherent to edge of orbits. Skin of a brownish color, with enlarged subcutaneous veins. Marked exophthalmos, the result of the outward pressure of tumors which extend to sub-conjunctival connective tissue; also smooth, distended swelling in region of temporal muscle which extended toward forehead, larger on right side. Nasal cavities and mouth normal. Ophthalmoscopic examination revealed extensive hemorrhagic retinitis of both eyes; papillæ red but not swollen; condition similar in each eye; vision, O.D., 20/xx; O. S., 20/xxx. Heart normal; urine sp. gr. 1010; reaction acid; a quantity of albumin, granular casts, and fatty epithelium; liver and spleen decidedly enlarged.

Blood examination showed a large increase of white blood-corpuscles. A small piece of growth removed from conjunctival surface was examined; it was soft, somewhat jelly-like, slightly transparent. Microscopical examination showed lymphoid cells crowded into connective-tissue meshes. This growth was, doubtlessly, analogous to leukæmic growths in other organs. Lymphatic glands of both sides of neck enlarged, and a large, painful, thickened tumefaction over sternum. Marked lassitude and debility. Temporary improvement derived from general sweats and acetate of potassium.

In May the patient's general condition was much worse; temporal and orbital tumors increased; liver and spleen greatly enlarged.

His condition grew steadily worse, and he died the last of October, delirium having existed for eight days. Leber believed this interesting condition to be the result of leukæmia. He, however, admits the possibility of the retinitis being due to the nephritis, but remarks that the extravasations extending to the periphery of the eye-ground are unusual in the latter disease. Unfortunately, an autopsy was refused.

Osterwald's case (reported *Archiv. Ophth.*, No. 27, 1881, p. 203). Patient, a boy aged four years, appeared at Göttingen eye clinic, May 25, 1881. Parents had always been healthy, both syphilis and hereditary disease being denied. The boy had measles at the age of two years, was pale and weak afterward, otherwise had been healthy. Soon after Easter, parents noticed a swelling in right upper eyelid, and later, a similar swelling in left, eyeballs protruding. When he appeared at the clinic the following symptoms were observed: Marked double exophthalmos. Both upper lids enlarged in all diameters, and under the skin of the left a network of bluish veins which extended to forehead and right temporal region. Right upper lid strongly pressed out by a hard, elastic tumor. This tumor began at orbital arch, to which it seemed attached, and extended from root of nose to outer angle of eye. In left upper lid was a similar, though smaller growth. In temporal region of either side was a smooth, round subcutaneous swelling of doughy consistency. Left conjunctiva and cornea normal; right conjunctiva injected and cornea ulcerated. Ophthalmoscopic examination showed left papilla cloudy, veins disturbed and tortuous; cornea interfered with examination of right retina. The presence of hemorrhage could not be determined on account of restlessness of patient. Vision not accurately ascertained, but the face of a watch could be seen. Small, hardened lymphatic glands on both sides of the neck. Complexion extremely pale and cachectic. Liver and spleen somewhat enlarged. Blood-count showed one white corpuscle to three or four red.

May 27, Professor Ebstein was called to the case. He mentions, in addition to the above symptoms, enlargement of inguinal lymphatic glands and nodules along costo-cartilaginous articulations of ribs. Blood fluid, watery, but not wanting in fibrin.

30*th*. Pulse rapid, feeble, some fever.

June 1. Slight fever.

2*d*. Temperature 38.3° C., epistaxis; evening temperature, 38.8° C.

3*d*. Evening temperature, 39.5°; bleeding of nose and mouth. Died at midnight. Autopsy by Professor Orth. A number of small tumors, firm, and of yellowish color, found in both pia and dura mater, which were distinctly of adenoid character. Heart contained large, yellow, firm coagula. Strong tendency to ecchymosis over surface of heart, pleuræ, and entire alimentary tract. Bronchial lymphatic glands normal; but cervical, axillary, inguinal and mesenteric glands were enlarged, as were liver and spleen. Both retinæ contained numerous small hemorrhages. Papillæ swollen and pale. On section the orbital tumors showed meshes of reticular tissue infiltrated by leucocytes. The meningeal nodules showed the same structure. Orth considered these leukæmic neoplasms of lymphadenoid variety, and a secondary result of the disease. The marrow of ribs and right femur was soft and tender, and of brownish-yellow color, characteristic of leukæmic marrow.

Fröhlich's case (reported in *Wiener med. Wochenschrift*, Nos. 7, 8, 9, 10, 1893). A man, aged twenty-five, a painter, was first seen July 27, 1892. Mother had history of frequent attacks of pulmonary hemorrhage; father and several sisters healthy. In youth the patient suffered from rhachitis, of which there are still marks on thorax. No history of syphilis.

May 1. Glands of axilla enlarged, associated with cough, expectoration, and dyspnœa; soon afterward other glands were involved, and upper lids of both eyes began to swell.

July 27. Tumors in lids, size of walnuts; fundus of left eye normal; right could not be examined on account of growth. Tonsils and pharynx normal; vocal cords pale and movable, with two dark-red subcordal tumors. Cervical and axillary lymphatic glands enlarged, also small swelling in middle of forehead, right humerus and right tibia. Examination of blood showed red corpuscles 3,570,000; white 137,000—one white to twenty-six red; hæmoglobin 70 per cent.

30*th*. Tracheotomy performed for the alarming dyspnœa, with complete relief. A sharp attack of pleuro-pneumonia terminated fatally November 24, 1892. Blood-count during the attack showed 2,882,353 red corpuscles; 8823 white—one white to 326 red. The writer attributes this apparent improvement in the condition of blood to the pleuro-pneumonia causing the destruction of lymphatic elements. The swellings in orbits and lymphatic glands, and the subcordal tumors, were greatly reduced, the latter so much that the voice before death was restored. A careful examination showed the tumors to be lymphomatous, and the reporter considered the disease to be pseudo-leukæmia.

Biesiadecki (*Jahrb. d. Ges. d. Aerzte*, 1876) describes a case of leukæmia with tumors of skin and enlargement of parotid

glands. He also cites cases related by Haltenhoff, Mikulicz, and Gordon Norrie, in which similar growths occurred in the upper lids and in the parotid and other salivary glands.

At the last Dermatological Congress, Paltauf and Riehl discussed the question of leukæmic skin tumors from a pathologico-anatomical standpoint.

DISCUSSION.

Dr. William F. Norris: These leukæmic growths of the orbit are very rare. There have been some cases published in German journals. Photographs of one case have been published, showing appearances similar to those seen in the photograph presented by Dr. Dunn. I have seen a number of cases of leukæmia, and examined the eye-grounds, but I have never seen the leukæmic growths in the orbit.

Dr. Dunn: In the three cases of lymphoid growths reported by Leber, Osterwald, and Chauvel, there was marked hemorrhagic retinitis extending to the periphery of the eye-ground, such as is common in leukæmic retinitis. It did not, however, seem to interfere extensively with vision in either of them.

SOME RESULTS OF A BACTERIOLOGICAL EXAMINATION OF THE PIPETTES AND COLLYRIA TAKEN FROM A TREATMENT CASE USED IN OPHTHALMIC PRACTICE, WITH THE EFFECTS OF INOCULATIONS.

A Preliminary Communication.

By G. E. de SCHWEINITZ, M.D.,
CLINICAL PROFESSOR OF OPHTHALMOLOGY, JEFFERSON MEDICAL COLLEGE; PROFESSOR OF OPHTHALMOLOGY, PHILADELPHIA POLYCLINIC;

AND

E. A. de SCHWEINITZ, Ph.D.,
CHEMIST IN THE BIO-CHEMIC LABORATORY, DEPARTMENT OF AGRICULTURE, WASHINGTON, D. C.; PROFESSOR OF CHEMISTRY, MEDICAL DEPARTMENT COLUMBIAN UNIVERSITY, WASHINGTON, D. C.

[Read April 3, 1893.]

The facility with which various fungi grow in many of the lotions commonly used in the treatment of ocular diseases, and particularly the proneness of the solutions of the alkaloids—cocaine, atropine, eserine—to accept this kind of contamination, has for a long time influenced the direction of much attention to the best methods of sterilization under these circumstances. True, these fungi and germs are not always, or not usually, pathogenic in the ordinary acceptation of the word; but the cases of wound infection—for example, after cataract extraction—from the use of unclean solutions, the microbic origin of toxic conjunctivitis (atropine, eserine and cocaine conjunctivitis), and the probability, as Philippson[1] declares, that certain ulcers of the cornea owe their origin to infected atro-

[1] Ueber Ulcus corn. serp., durch Einträufelung septischer Atropinlösungen hervorgerufen. Hosp. Tid. R. iii., 1885; quoted by Franke, Archiv f. Ophthal., Bd. xxxvii. Abth. ii. 73.

pine drops, furnish grounds for the exercise of every proper precaution to secure clean fluids, bottles and pipettes.

The germs, as Franke has shown, may be present in the bottles and pipettes, or as Davidson[1] has demonstrated, in the distilled water (micrococcus aquatalis, etc.); again, they may come from the surrounding air; or, finally—and this, as Franke insists, presents the greatest difficulty to overcome—they may be introduced into the lotions by the surgeon himself when, for example, the end of the pipette has accidentally touched the conjunctiva or the eyelashes, and thus carried into the bottle some of the micro-organisms so commonly present in these structures.

Franke, to whose research we have referred, gives a very interesting *résumé* of the various procedures to secure sterilization under these circumstances. From the time that Kroemer added to solutions of the alkaloids salicylic acid 1 : 400, boric acid 4 : 100, and carbolic acid 1 : 1000, which even in this strength was found sufficient to prevent the growth of fungi, up to the present date a number of methods have been practised which may be summarized as follows: Sterilization by heat, by the addition of an antiseptic, by the combination of these two methods, and by the chemical synthesis of alkaloids with antiseptic acids.

Particular attention has been directed to cocaine. Sattler, for example, was accustomed to prepare a solution of this alkaloid by adding to it a 1 : 5000 solution of bichloride of mercury, which later was changed to a 1 : 10,000 solution upon the strength of some investigations made by Herrnheiser. The same method of preparing cocaine is extensively used by many surgeons.

Eversbusch boiled the solution preparatory to an operation, a procedure which sterilizes the fluid itself, but does not prevent the entrance of bacteria from the surrounding air.

Hirschberg, with characteristic thoroughness, first sterilized his solution in the ordinary manner in a sterilizing apparatus, and then added to it sublimate 1 : 5000.

[1] Berlin klin. Wochenschr. 1888, No. 35; quoted by Franke, loc. cit.

Finally, an attempt to secure an antiseptic drug has been made by combining eserine and cocaine with salicylic acid in the form of salicylate of eserine and salicylate of cocaine, a pharmaceutical experiment at one time indorsed by Galezowski and Petit.

As there seemed objections to some of the procedures, and uncertainties in regard to others, Franke has reviewed the whole subject, endeavoring to discover the simplest and safest method of sterilizing eye lotions, and has devoted himself to the sulphates of atropine and eserine and the hydrochlorate of cocaine, the first in 1 per cent. and the second in 2 per cent. solution.

The most important conclusions given by the author at the close of his paper are here summarized: Chemical disinfection of "eye drops" is a method in general to be preferred to a sterilization by heat, because by the latter procedure the lotions are not protected from the micrococci which may fall into them from the surrounding air. The drugs which may be employed for this purpose are sublimate 1 : 5000 and 1 : 10,000, oxycyanide of mercury 1 : 1000 to 1 : 1500, resorcin 1 per cent, carbolic acid $\frac{1}{2}$ per cent., boric acid 4 per cent. with 1 per cent. of carbolic acid, Panas's fluid, and thymol in the form of thymol water, or chloroform water. The last two substances have the disadvantage of producing smarting and burning of the conjunctiva. In their behavior toward the staphylococcus pyogenes flavus and Michel's trachoma coccus, sublimate 1 : 1000, oxycyanide of mercury 1 : 1000, and thymol water, surpass the others.

It is not possible, however, for ophthalmic purposes to use these lotions in a strength sufficient to obtain an absolutely antiseptic action, but in general it is safe to add a portion of sublimate lotion (1 : 10,000) to atropine and cocaine solutions, in order to render them aseptic for a space of from one-half to one hour.

Sterilization alone, of atropine and cocaine solutions, when they are used in operative work, without the addition of an antiseptic, is not nearly so satisfactory as a combination of the two methods.

The addition of a 1 : 10,000 sublimate lotion to an atropine solution prevents atropine conjunctivitis.

In addition to these precautions, it is recommended that the bottles and pipettes be sterilized by boiling and mechanical cleaning. In short, two methods of sterilization are practicable, namely, the addition of a chemical substance and the use of heat, and even Franke does not feel quite safe unless he employs both.

Pergens,[1] after reviewing the various methods of sterilization, and pointing out the advantages and disadvantages of each, suggests that hypodermic tablets, which have been carefully manufactured and well dosed, shall be prepared. These are dissolved in sterilized water and poured into bottles of the capacity of three to five grammes. Each patient is treated with a different tube, which is then plunged in the sublimate solution, where it remains for several hours. These pipettes are washed with water and sterilized with steam, because he believes that they are the principal source of infection.

Heat alone, however, is entirely sufficient, if proper precautions are taken that the surrounding bacteria shall not fall into the fluid, and if a suitable method be employed for heating the fluids. To this end Stroschein[2] has devised a new bottle, or rather a combination of flask and pipette. He has had blown-glass bottles constructed with droppers which may be directly exposed to the flame, thus rapidly sterilizing the bottle and its contents. The pipette has two conical ends, and is introduced into the neck of the bottle point upward, the rubber head being removed before the reversal. If the collyria are to be boiled the small tube gives free vent to the steam, which, passing through it, at the same time sterilizes it. Experiments have shown the inventor that the collyria need to be boiled only three or four minutes to render them perfectly sterile, and for the point of the tube, which is directed upward during the ebullition, likewise to become sterile. The loss of water which is produced by a

[1] Annales d'Oculistique, December, 1891.

[2] Archiv. f. Ophthalmologie, Bd. xxxviii, Abth. 2, p. 155.

boiling of three or four minutes is about one cubic centimetre, hence, if concentration of the solution is to be avoided, fifteen drops of water should be added before the boiling.

A number of so-called antiseptic droppers have been devised, one well-known pattern consisting of a combination of the dropper and glass stopper in a single piece of glass. All of these devices are ingenious, but do not secure the introduction into the eye of an aseptic fluid, because, as has been proved over and over again, the water in which the drug is dissolved may not be clean, and solutions of the alkaloids, with the possible exception of eserine, even when freshly prepared, will usually yield a growth of fungi and various micrococci in culture media.

Although the question of infection and sterilization of eye lotions has so frequently and so thoroughly been investigated, we desire to present the results thus far obtained in a research the object of which was to ascertain, purely for our own satisfaction, the condition of the fluids, pipettes, and bottles contained in a case which had been much used in the treatment of various ocular disorders, although never in operative work, and with which no special precautions had been taken. The examinations are not complete; but they have developed some points of interest, and these may be briefly stated.

The following fluids were taken from the bottles and carefully placed in sterilized flasks and suitably corked: Boric acid, 15 grains to the ounce of distilled water; bichloride of mercury 1 : 10.000; nitrate of silver 1, 5 and 10 grains to the ounce respectively; sulphate of atropine, 4 grains to the ounce; hydrobromate of homatropine, 8 grains to the ounce; hydrochlorate of cocaine, four per cent.; sulphate of eserine, 1 grain to the ounce; pyoktanin 1 : 1000; and Gruebler's fluorescine, two per cent.

The pipettes which had been used in these fluids, and with two exceptions in them only, as each one is attached to its own bottle by a special arrangement, were also placed in carefully sterilized flasks and examined.

The droppers were tested in the following manner: Thor-

oughly sterilized distilled water was drawn up into each pipette until the latter was three-quarters full, and then forced out by the rubber bulb and allowed to drop into potato tubes, peptonized beef broth, and agar-agar. The tubes were then placed in the incubator and examined at the end of forty-eight hours.

The cocaine and eserine droppers treated in this way gave, in forty-eight hours, growths on all three of the culture media, which upon examination proved to be a mixed culture of long and short bacilli and micrococci.

After two weeks a growth was noticed on the potato culture from the fluorescine pipette; but it is not certain that this was not the result of a contamination.

Other droppers tested in exactly the same way, namely, those from the atropine, homatropine, boric acid, and pyoktanin solutions and an unused dropper, yielded no growth whatever.

The lotions themselves were treated by transferring to the culture media—potato, peptonized beef broth, agar-agar—by means of a platinum loop, drops of the liquid to be examined. The cocaine, boric acid, atropine, and homatropine lotions developed growths on potato, peptonized beef broth, and agar-agar. Drop inoculations from the fluorescine were negative, but when one-half drachm was used an abundant fungus growth appeared. The other solutions—namely, eserine, nitrate of silver, pyoktanin, and biehloride of mercury—developed nothing at all.

The pathogenic effects of the cultures so far obtained, though not pure, were next tried. The agar-agar cultures were the source of the material for injection, and an emulsion of the surface growth and the condensation water of the culture was used, one-tenth of a cubic centimetre for each injection. The injections were made by means of a hypodermic syringe into the anterior chamber of a rabbit's eye, care being taken not to injure the lens. The cultures from which injections were made were from the boric acid lotion, cocaine, fluorescine (dropper), homatropine and eserine (dropper).

The eserine growth (pipette) caused in the eye of the rabbit a slight inflammation of the iris which disappeared in two days.

The growth from homatropine produced a mild iritis which lasted for four days.

The culture from the fluorescine dropper provoked a slight iritis associated with a moderate keratitis which disappeared spontaneously in eight days.

The cultures from the boric acid and the cocaine, the first derived from the solution and the second from the pipette, produced a violent hypopyon-keratitis and purulent iritis.

Rabbits were then taken and the corneæ abraded with a sterilized needle. The boric acid and cocaine cultures were dropped on eyes thus prepared, and rubbed over the abraded surfaces. The result was a slight ciliary injection in the region of the abrasion and a moderate haze in the cornea surrounding it; this condition lasted for two or three days, and was more pronounced than the inflammation produced by a simple abrasion of the cornea without the application of the germs.

In order to control the injections in the anterior chamber, the eserine solution, which had not produced a growth upon the culture media, was injected, without results; hence the hypopyon-keratitis developed was not the effect of the fluid, but of the bacilli and fungi which it contained. Further control experiments with negative results were made by the injection of sterile culture liquids.

The cultures from the boric acid and cocaine show under the microscope long and short bacilli and micrococci; proteus vulgaris and bacterium termo (Vignal) have been isolated. The bacilli have the general appearance of the ordinary water bacilli. Sufficient time has not yet elapsed to separate them in a pure culture. The homatropine growth and the one from the eserine dropper showed short fat bacilli. The atropine and fluorescine (dropper) cultures are the ordinary fungi.

The experiments in detail, with the daily appearance of the eyes, follow:

November 30, 1892. Gray rabbit No. 1; weight four pounds. Received $\frac{1}{10}$ c.c. of the culture obtained from the eserine dropper.

December 1. The eye was very slightly injected and iris hyperæmic.

2*d*. Eye entirely well.

November 30, 1892. Gray rabbit No. 2; weight four and one-fourth pounds. Received an injection in the left eye of $\frac{1}{10}$ c.c. culture from the fluorescine dropper.

December 1. Centre of the cornea cloudy.

2*d*. Cornea still clouded; the bloodvessels of the iris decidedly injected.

3*d*. Still a slight inflammation of the iris; apparently a little lymph in pupillary space, but the cornea no longer cloudy.

5*th*. Iris still slightly inflamed; lymph had almost entirely disappeared.

6*th*. Slight iritis.

7*th*. Slight iritis, but better.

9*th*. Still a very slight inflammation.

13*th*. Eye entirely well.

November 30, 1892. Rabbit No. 3; weight four pounds. Received $\frac{1}{10}$ c.c. from culture growth of homatropine solution.

December 1. Iris a little inflamed and a slight cloudiness in pupillary space.

2*d*. Eye still inflamed.

3*d*. Inflammation subsiding.

5*th*. Eye almost normal.

7*th*. Slight iritis.

9*th*. Slight iritis.

14*th*. Eye perfectly well.

November 30, 1892. Brown rabbit No. 4; weight four pounds. Received $\frac{1}{10}$ c.c. of culture from cocaine dropper.

December 1. Cornea very cloudy and inflamed.

2*d*. Eye swollen, inflamed, suppurating, and hypopyon.

3*d*. Extensive iritis.

7*th*. Extensive purulent iritis.

9*th*. Eye about the same as on December 7th, except that the bloodvessels in the cornea had become very much more prominent.

14*th*. Eye in the same condition as on December 9th.

February 7, 1893. Eye removed. There had been practically no change in its appearance since December 14th.

November 30, 1892. Rabbit No. 5; weight four and one-half pounds. Received an injection of $\frac{1}{10}$ c.c. of culture from the boric acid solution.

December 1. Cornea clouded.

2*d*. Still slightly clouded, and the iris hyperæmic.

3*d*. Extensive iritis and keratitis.

5th. Eye apparently better.

6th. Relapse; severe iritis and keratitis.

7th. Still severe kerato-iritis.

9th. Same as on December 6th.

13th. Entire cornea infiltrated; severe hypopyon-keratitis.

February 7, 1893. Eye removed for section; apparently the same condition as on December 13th.

December 13, 1892. Gray rabbit No. 6; weight four pounds. Received $\frac{1}{10}$ c.c. in left eye, of culture obtained from cocaine solution. This solution when first tested in October had shown no growth, but when cultures were made again in December, a decided growth was developed upon agar, consisting of long and short bacilli.

February 7. When the eye was removed its appearance was almost identical with that of the boric acid and other cocaine eye, viz., suppurative kerato-iritis.

January 7, 1893. Rabbit No. 7; weight three and one-half pounds. Cornea of right eye was scraped with a sterile knife blade, and some of the boric acid culture dropped on the abraded spot and rubbed over it.

9th. A slight injection of the bloodvessels in ciliary region just above the point where surface was abraded. A small cloudy spot, the size of a pin-head, formed about the centre of the line of abrasion.

12th. Eye normal.

January 7, 1893. Maltese rabbit No. 8; weight three pounds. Surface of cornea abraded and a few drops of the culture from cocaine solution rubbed in.

8th. Slight reddening in ciliary region and cloudiness in cornea.

12th. Faintest cloudiness in cornea; otherwise quite well.

February 9. Eye normal.

January 7, 1893. Black Angora rabbit No. 9; weight four pounds. Surface of cornea scraped and a few drops of culture from cocaine dropper rubbed in.

9th. Injection of bloodvessels in ciliary regions above the point where the surface was scraped.

12th. Inflammation has subsided; a very slight cloudiness above where the surface was abraded.

This experiment of scraping the cornea was repeated again on January 21st, with exactly the same results as those recorded. Simply scraping the cornea produced a slight injection of the bloodvessels in the ciliary regions; not so much, however, as when the cultures were subsequently applied.

A summary of the results, arranged in a tabular manner, follows:

PIPETTES.

Unused pipette.	No growth.		
Cocaine pipette.	Growth in potato, agar-agar, and beef broth.	Same germs as those found in cocaine solution; inoculation caused purulent irido-choroiditis.	Rubbing abraded cornea with culture produced moderate ciliary injection and slight clouding of cornea.
Fluorescine pipette.	Growth on potato.	Inoculation caused slight iritis.	
Atropine pipette.	No growth.	Pipette had been frequently cleansed with sublimate lotion.	
Pyoktanin pipette.	No growth.		
Homatropine pipette.	No growth.		
Eserine pipette.	Growth on all three culture media.	Slight iritis caused by inoculation which speedily disappeared.	
Boric acid pipette.	No growth.	This pipette had often been used with a sublimate lotion.	

FLUIDS.

Boric acid lotion.	Active growth on all three culture media.	Inoculation into anterior chamber produced purulent irido-choroiditis.	Rubbing abraded cornea with culture produced moderate ciliary injection and slight clouding of cornea.
Atropine lotion.	No growth at first; one month later active growth.	The growth, a fungus, was not used in inoculation, as it was of same nature as that obtained from the fluorescine dropper.	
Homatropine lotion.*	Growth on all three culture media.	Inoculation produced slight iritis which disappeared in fifteen days.	
Cocaine lotion.	No growth at first; two months later active growth on all three culture media.	Inoculation produced purulent irido-choroiditis.	Rubbing abraded cornea with culture produced moderate ciliary injection and slight clouding of cornea.
Fluorescine lotion.	Abundant fungus growth.	Same growth as from pipette not used in inoculation.	
Bichloride lotion.	No growth.		
Nitrate of silver lotion	No growth.		

The two eyes which had suffered most severely from the injections of the cultures, viz., from the cocaine and the boric acid, were removed and submitted to microscopic examination. As the lesions are practically the same in each, one description will suffice. The iris is infiltrated with leucocytes, and there is a large layer of pus on its posterior surface, in some specimens practically filling the posterior chamber. The ciliary body and choroid are densely infiltrated with darkly stained corpuscles, the

retina is detached and infiltrated, and the papilla inflamed. In brief, there is purulent irido-choroiditis with secondary involvement of the retina and optic nerve.

We are indebted to Dr. William M. Gray for preparing these sections.

These experiments, in general and as far as they go, confirm the oft-repeated observation that solutions of the alkaloids contain mould-fungi, *e. g.*, aspergillus glaucus, saprophytic bacteria, and sarcina lutea. They also confirm the observations which have been recorded, that these solutions, when they have been some time in use, and when proper precautions are not taken to sterilize them, may be contaminated with pathogenic micro-organisms which are capable of producing by inoculation into a normal eye a purulent irido-choroiditis.

Referring for a moment to the particular results, it is interesting that one of the two most virulent cultures was obtained from the boric acid lotion, a culture which evidently contained pathogenic germs. The source of these micro-organisms may, perhaps, be explained by Franke's idea that they are introduced into the fluid by reason of the pipette coming in contact with the cilia or conjunctiva of an eye which contains pathogenic bacteria, and it is likely that this lotion was frequently used for irrigating inflamed conjunctivæ. It serves as an illustration of the well-known fact that boric acid is an antiseptic substance of very indifferent power, the minimum degree of concentration in a watery solution in which it is reliable, according to Miquel, being 1 : 13. In the solution with which we experimented the concentration was 1:32.

It is further interesting to note that the solution of eserine yielded no growth upon the culture medium, and that when it was injected into the anterior chamber it proved to be innocuous. This is somewhat in accord with some of Franke's observations, who found in a few inoculations from freshly prepared eserine solutions, and also from those which were several days old, that the tubes remained free from growths. The eserine pipette yielded a growth of long and short bacilli and micrococci, consequently its contamination must have come from outside sources which did not gain entrance into the fluid in the bottle.

If it be assumed that the culture from the fluorescine dropper was not due to a contamination (it did not appear until two weeks after the potato tube was inoculated), it is worthy of remark that it produced a slight iritis when injected into the anterior chamber. Fluorescine solution has been much used in recent times to demonstrate the extent of corneal ulcers, to locate small foreign bodies, and to expose, by virtue of its power to color green those portions of the cornea which are deprived of superficial epithelium, any loss of substance in this membrane, whether caused by injury or by disease. Hence there is a possibility that by this means micro-organisms might be conveyed to the eye and convert a simple abrasion into an unhealthy ulcer. In our experiment the fluid itself was at first apparently sterile, but subsequently inoculations gave an abundant fungus growth of the same character as that obtained from the dropper.

That mere contact of the cultures with an abraded cornea is unlikely to produce serious inflammatory reaction or purulent infection is evident from our last four experiments, in which the infecting agent was rubbed into a wound of the cornea made with a sterilized knife. Ciliary injection and slight corneal infiltration occurred, which subsided in a few days without treatment, but destructive keratitis did not supervene in any case, although the same cultures injected into the anterior chamber caused purulent irido-choroiditis.

Hence we have a reason for the comparative rarity of untoward results from non-sterile solutions that are so frequently (and almost of necessity) used in eyes manifesting all manner of lesions, but in which the anterior chamber is not opened.

They also furnish additional evidence that unclean solutions which find their way into the anterior chamber, *e.g.*, after an operation, are capable of speedily originating a destructive inflammation of the uveal tract, terminating in panophthalmitis, and emphasize the importance of securing perfect sterilization of *any* lotion, especially of cocaine, which is to be used in a case requiring corneal section; showing also that boric acid solution which is not freshly prepared (gr. xv to f℥j) may be the medium of the most virulent contamination.

THE ETIOLOGY OF MEMBRANOUS RHINITIS (RHINITIS FIBRINOSA).

By A. C. ABBOTT, M.D.,

FIRST ASSISTANT IN THE LABORATORY OF HYGIENE, UNIVERSITY OF PENNSYLVANIA; FELLOW OF THE COLLEGE OF PHYSICIANS, PHILADELPHIA, ETC.

From the Laboratory of Hygiene, University of Pennsylvania.

[Read May 3, 1893.]

During the past year or two the condition of the nasal mucous membrane commonly known as membranous rhinitis has occasionally claimed the attention of bacteriologists, the result of whose studies has been to show that a fair proportion of cases of this relatively rare disease are of a diphtheritic character, and that diphtheria bacilli possessing their full virulence can often be isolated from them.

These observations, in connection with the fact that this affection is commonly looked upon as non-communicable, call for certain modifications in the care of these cases—modifications concerning less the treatment than the isolation of these patients; for, in so far as I am prepared to say, the treatment received by them is that which might be, and ofttimes is, profitably employed in typical pharyngeal and laryngeal diphtheria.

But, as has been said, these cases are usually not considered of a dangerous character, and are practically never isolated nor prevented in any way from mingling, and, indeed, often coming into intimate contact with healthy individuals.

They are, as a rule, seen in dispensary practice; they rarely or never present constitutional symptoms, and for these reasons

do not receive the attention that their gravity indicates. I have no positive evidence that diphtheria has ever been contracted from one of these cases, but from what we now know of them it is not improbable that this has more frequently happened than has hitherto been supposed. The comparative rarity of this disease necessarily limits the opportunity presented for its study, but the observations that have been made by reliable authorities point so directly to the diphtheritic nature of many of these cases that, where possible, it seems advisable to insist upon the bacteriological examination of all pseudo-membranous conditions of the nasal cavity that are not directly and positively traceable to other causes.

As results of such studies as have been made, Concetti,[1] in five cases of pseudo-membranous rhinitis obtained from two of them by culture methods, the virulent diphtheria bacillus; in two others, a direct history of infection from one case to the other, with, in one of these latter cases, subsequent diphtheritic paralysis; while in the fifth case there was a secondary appearance of a membranous condition in the larynx. In all five cases the course was chronic, and, with the exception of the last case, was limited to the nose.

Stamm[2] examined, by bacteriological methods, four cases of typical membranous rhinitis, and found in all of them the virulent bacillus diphtheriæ.

Baginsky[3] describes two cases of membranous rhinitis, both of which ran a benign course, and from both of which the bacillus diphtheriæ was obtained by culture methods.

In his elaborate researches upon diphtheria and allied pseudo-membranous inflammations, Park[4] gives the results of analyses of six cases of the disease. In all these cases the Klebs-Löffler bacillus was present—in five of them in relatively large numbers. As is usually observed, these cases ran a benign course, and no history of infection was obtained from any of them.

[1] Concetti, Luigi: Archivio Ital. di Laringologia, 1892, anno xii; reference from Centralblatt für Bakteriologie u. Parasitenkunde, 1892, Bd. xii, p. 673.

[2] Stamm, C.: Archiv für Kinderheilkunde, Bd. xiii., Heft 3.

[3] Baginsky: Berliner klin. Wochenschrift, 1892, No. 9.

[4] Park: Medical Record, July 30 and August 6, 1892.

During the past nine months we have had an opportunity of examining three cases of membranous rhinitis, in all of which the disease was limited to the nasal cavity. For the first case we are indebted to Dr. B. Alexander Randall, of this city, who saw the child in the dispensary service at the Children's Hospital, and kindly permitted us to make an examination of a portion of the membrane removed by him. The clinical history of the case is not complete, for the child disappeared, and did not return to the dispensary. The bacteriological examination of this case, which was made by Dr. Ghriskey, revealed the presence of numerous colonies of bacillus diphtheriæ which, when inoculated into guinea-pigs, caused their death with all the usual pathological lesions commonly produced by this organism in the bodies of these animals. There was no apparent diminution of virulence, and no deviation in the morphology of these organisms, nor in their cultural peculiarities, from those common to diphtheria bacilli obtained from undoubted cases of primary diphtheria.

The two remaining cases are especially interesting, from the fact that they occurred in two children in the same family, and doubtless represent an example of direct infection from one child to the other. For these cases I am indebted to Dr. Walter J. Freeman, of Philadelphia, who has kindly supplied me with the following histories.

The first of the two cases was a girl (M. W.) aged seven years, who had had trouble with the nose for six or seven weeks prior to examination. It began as a cold; there was no headache, no fever, no sore-throat. Both nares were stopped up at the time of examination by thick membranous deposits, which were so adherent that in removing a piece, epistaxis of such a degree occurred that it became necessary to tampon the anterior portion of the nose for twenty-four hours. Ten days afterward the child appeared at the clinic; the membrane had entirely disappeared under treatment, and she was pronounced well.

Three days after this first child came to the dispensary, the younger sister, aged two years, was brought by the mother, who stated that one week prior to that date the child had vomited, and on the evening of that day had a fever; was without appetite; was restless at night; the breathing heavy and the nose "stopped up." Examination of this child in the dispensary

revealed the presence of membrane in both nares. There was no sore-throat nor had there been any, and the temperature was 99° F.

On April the 11th, twelve days after this child first appeared at the dispensary, I had an opportunity of making a bacteriological examination of the deposit in the nose. At this time both nares contained membrane, but not sufficient to plug them completely. The breathing of the child was principally through the mouth; there was no sore-throat, and otherwise she showed no evidence of any abnormal condition. The result of the bacteriological study of the membrane from the older sister revealed the presence of the Klebs-Löffler bacillus in large numbers. Cultures from single colonies were not only identical in all morphological and cultural respects with Löffler's bacillus as obtained from a genuine case of diphtheria, but also, like it, when inoculated subcutaneously into guinea-pigs, caused their death in less than forty-eight hours, the autopsy revealing the pathological lesions characteristic of these inoculations.

From the younger sister, cultures of bacilli morphologically and biologically identical with Löffler's bacillus were obtained, but when inoculated into guinea-pigs they were found to be of much lower degree of virulence than those obtained from the older sister. They did not cause the death of the animals, but produced only a local swelling and a very temporary indisposition, from which they recovered. In confirmation of Park's observation, the cultures obtained upon the day of examination from the older sister, and which were, as stated, virulent at that time, were found after thirty days not only devoid of virulence, but indeed, devoid of vitality, and it was impossible to get them to grow again even under the most favorable conditions of cultivation. Except for the absence of pathogenic properties, the bacilli obtained from the latter case seen by me could not, by any of the means usually employed, be differentiated from the genuine virulent bacillus diphtheriæ. Two guinea-pigs were inoculated with relatively larger amounts of cultures from two distinct single colonies on the original tubes from the nose, but, as stated, they did not succumb to the operation. The absence of virulence from the cultures of the

latter case is not entirely surprising in the light of Park's observations, who found that in the six cases of rhinitis examined by him, all of which ran a benign course, the bacilli obtained in culture were all of a low degree of virulence, though it is interesting, in view of the probability of infection from one sister to the other in my cases, that in the one there should have been virulent and in the other non-virulent organisms, otherwise identical. Whether this was due to the prolonged efforts of the tissues in resisting invasion, gradually depriving the organisms of their disease-producing powers, or to the repeated application of disinfecting solutions to the surfaces upon which they were located, it is impossible to say; but we know with certainty that particular pathogenic organisms subjected for only a relatively short time to the action of disinfectants, show when not completely destroyed, the effects of the treatment by reduction in their pathogenic activity.[1]

In neither of the latter two cases seen by me have I been able to obtain a history of any marked constitutional disturbance that would lead one to suspect the presence of a grave condition.

It is very interesting, but a little confusing, to find the same organism when located at different points giving rise to such essentially different clinical conditions as are seen when its seat of activity is the tissues of the fauces, as in diphtheria, and the tissues of the nasal cavity, as in membranous rhinitis. In the latter affection constitutional expression is the exception, and the course of the disease is usually more or less chronic, while the stages through which the typical diphtheria patient passes are too well known to us all to require rehearsal here.

The differences in virulence that are observed in the organisms obtained from cases of membranous rhinitis, and less frequently from diphtheritic patients, may serve, in part at least, to explain the variations that are seen in the intensity, not only of the nasal form of the disease, but in true diphtheria as well. The bacilli present in genuine diphtheria are, with but few ex-

[1] See paper by the author, "Corrosive Sublimate as a Disinfectant against the Staphylococcus Pyogenes Aureus," Johns Hopkins Hosp. Bull., No. 12, April, 1891.

ceptions, highly pathogenic for certain animals, and while this is often true of membranous rhinitis, it is, nevertheless, not unusual to find organisms that are constant in morphological and cultural identity with the bacillus diphtheriæ, but which differ from it in the degree of their virulence, at times possessing this property, but diminished in intensity, and not infrequently being entirely devoid of it.

These observations undoubtedly offer another argument in support of the opinion advanced by Roux and Yersin,[1] myself,[2] and others, that the virulence of the bacillus diphtheriæ may be observed to fluctuate in the degree of intensity—at one time possessing this property in a high degree, at another presenting a decided attenuation, and not infrequently a complete absence of pathogenic power; and that the virulent bacillus diphtheriæ and the so-called pseudo-diphtheritic bacillus are one and the same organism, the former being most commonly concerned in the production of a grave condition, associated with constant, or nearly constant, pathological and clinical expressions, while the tissue-changes and constitutional manifestations that present themselves as a result of the activities of the latter may vary with the degree of its disease-producing power. This hypothesis, of course, finds its most favorable application to those cases of rhinitis or other mild diphtheritic inflammations the clinical history of which would *a priori* almost exclude the probability of highly virulent organisms being concerned in their etiology.

With regard to the lack of constitutional disturbance in those cases of rhinitis running the usual benign course in which virulent diphtheria bacilli are present, it must be borne in mind that not infrequently cases of genuine laryngeal or pharyngeal diphtheria are seen from which organisms possessing their full virulence can be isolated and from which all constitutional symptoms indicative of grave disorders are absent; a condition that, in the present state of our knowledge, we feel justified in referring to an exaggerated or unusual de-

[1] Roux and Yersin: Annales de l'Institut Pasteur, 1892, tome iv.

[2] Abbott: Johns Hopkins Hospital Bulletin, October and November, 1891, No. 17.

gree of resistance offered to the invading organisms by the tissues of the individual affected; a condition of the tissues formerly vaguely spoken of as "vital activity," but now generally recognized upon experimental evidence as a potent factor in repelling the inroads of disease-producing organisms.

That the organism that we have been accustomed to know as the pseudo-diphtheritic bacillus, though incapable of destroying the life of guinea-pigs into which it has been inoculated, does, nevertheless, possess the power of causing in these animals limited localized tissue-changes indistinguishable, save in degree, from those produced by the typical virulent bacillus, is borne out by microscopic examination of the tissues at the seat of inoculation in animals into which these non-virulent forms have been introduced, but which have not succumbed to the operation. For example, of the two guinea-pigs inoculated subcutaneously with cultures from the last case mentioned by me, neither of which succumbed, and in both of which only local tumefaction occurred, one that had gone for ten days with no apparent constitutional disturbance was killed after this date. The autopsy revealed, at the seat of inoculation, a yellowish area marked by numerous hemorrhagic points and surrounded by a very limited area of œdema of not over 0.5 c.m. in extent. This yellowish area was firmly adherent to the overlying skin and extended into the substance of the abdominal wall. The peritoneum beneath was slightly reddened and the inguinal gland on the side inoculated was very slightly enlarged and reddened. Upon opening the peritoneal and pleural cavities no fluid was present; the retro-peritoneal lymph-glands were neither enlarged nor reddened. The intestines were normal; the kidneys and liver were normal; the spleen was dark in color, otherwise normal; the adrenal bodies were small and pale, and upon section contained a brownish fluid; there was no excess of fluid in the pericardium, and the lungs were normal. Subcutaneous lymph-glands over the body, except the inguinal nearest the seat of inoculation, showed no change.

Culture media (Löffler's blood-serum and agar-agar) inocu-

lated from the seat of inoculation, the blood of the heart, the liver, and the spleen, remained sterile.

Microscopic examination of sections of the seat of inoculation, hardened in alcohol and stained with fuchsin, revealed a condition in all essential respects identical with that described by Welch and Flexner[1] at the seat of inoculation of animals dead after the subcutaneous introduction of virulent cultures of this organism. There was the same emigration and destruction of leucocytes, the nuclei of many of which were conspicuously disintegrated. The nuclei of the fixed cells were also seen to be undergoing destruction. Many of the muscle fibres were hyaline, and at places were seen to be penetrated by leucocytes. When stained by Weigert's fibrin stain a delicate network of fibrin could easily be made out.

In short, the tissue changes were in all respects, save for a diminution in degree, identical with those produced by the virulent bacillus diphtheriæ.

For this reason we are inclined to the opinion that the term "pseudo-diphtheritic bacillus"—as applied to an organism in all respects identical with the genuine diphtheritic bacillus, save for its inability to *kill* guinea-pigs when inoculated subcutaneously—is a misnomer, and that it would be more nearly correct to designate this organism as the attenuated or non-virulent diphtheritic bacillus, reserving the term "pseudo-diphtheritic" for that organism or group of organisms—for there are probably several—that is sufficiently like the diphtheritic bacillus to attract attention, but is distinguishable from it by certain morphological and cultural peculiarities, aside from the question of virulence.

It is a well-known fact that many pathogenic organisms—conspicuous among these being the diplococcus pneumoniæ, the staphylococcus pyogenes aureus, and the group of so-called hemorrhagic septicæmia organisms—undergo marked variations in the degree of their pathogenic properties; and yet these organisms, when found either devoid of this peculiarity,

[1] Welch and Flexner: Johns Hopkins Hospital Bulletin, August, 1891, No. 15.

or possessing it to a diminished extent, are not designated as "pseudo" forms, but simply as the organisms themselves, the virulence of which, from various causes, has been modified.

Another interesting point to which Park calls attention is the peculiar morphological variations that he observed in the organisms obtained by him from the six rhinitis cases that came under his observation. He states that the bacilli from the agar-agar cultures were small and often pointed, while those from the blood serum and broth cultures were long and slender, with swollen ends. We have repeatedly noticed this condition not only in the cultures from the rhinitis cases, but from cases of genuine pharyngeal diphtheria as well. We have recently been engaged in a series of experiments upon this point, and, though they are not as yet complete, the results thus far obtained are, we think, of sufficient importance to justify mention here. The morphology of the organism while usually described as conspicuous for its irregularity, is, nevertheless, relatively constant under one and the same condition of cultivation. That is to say, the individuals comprising the growth on blood serum (Löffler's mixture) are very long, irregular, sometimes clubbed, sometimes pointed at the ends, and are conspicuous for the irregular manner in which they take the staining; while the growth of the same organism on glycerin agar-agar is far less voluminous. and the individuals composing it are, as a rule, short, often not over from one-sixth to one-fifth as long as the forms seen on blood serum; they are pointed, curved, clubbed, spindle-shaped, lancet-shaped, stain more uniformly and, without exception, possess pale transverse markings, when stained with Löffler's blue, that give to them the appearance of being made up of very short segments. (See illustration.) It is interesting to note the transition from the one form to the other when transferred from the one to the other culture medium. We have repeatedly isolated from cases of diphtheria the diphtheritic bacillus by Löffler's blood-serum method and continued its pure cultivation upon blood serum through five and six generations, in

all of which practically only the long, irregular, imperfectly-staining rods could be seen, but as soon as a cultivation upon glycerin agar-agar was made from one of these generations, without a single exception, only the short segmented forms previously described would develop. Similarly, if the cultures

Bacillus diphtheriæ. *a*. After twenty-four hours' growth on six per cent. glycerin agar-agar, at 37° C. *b*. After twenty-four hours' growth on Löffler's blood-serum, at 37° C. Magnified in both cases about 1500 diameters.

were obtained from the throat upon glycerin agar-agar and continued through a number of generations on this medium, the transition from the short segmented to the long irregular threads was seen within twenty-four hours after transferring them to the blood-serum mixture.

All these long irregular forms have hitherto been referred to collectively as involution or degenerate conditions of this organism, but I cannot reconcile the voluminous development within twenty-four hours, as seen on blood serum, the most favorable medium for the growth of the diphtheria bacillus,

with the existence of a diseased condition of the individuals of which the growth is composed. We have made these observations upon cultures obtained from five different cases of genuine primary diphtheria, from two cases of membranous rhinitis, and upon one culture of the so-called pseudo-diphtheritic bacillus, and find them to hold for all. To which constituent of the culture media the peculiar and sudden transitions are due we cannot definitely say at present, but as the work is still in progress we hope to speak positively upon this point in the near future. These points of difference are not alone of technical interest, for it is important for the clinician who employs bacteriological methods in the study of his cases to realize that, at least in the case of diphtheria, the morphology of the organism causing it varies with the medium employed for its isolation and cultivation, and, unless he is familiar with the appearances presented by it under the varying conditions of environment, it is possible for error in diagnosis to arise, even though the manipulation of the case and the preparation of the cultures may have been practised with all the precautions necessary to success.

RED CHROMIDROSIS (?)

By M. B. HARTZELL, M.D.

[Read May 3, 1893.]

The disease of which the three following cases are examples is not a true chromidrosis, the sudoriparous glands probably being unaffected, or, at most, playing only a secondary part in its production; but as this term is employed to designate the malady in most text-books upon dermatology, it is here retained to avoid possible confusion.

Case I.—Mrs. S., thirty-five years old, blonde, with red hair, nervous temperament; suffers from dysmenorrhœa; sterile, although married for fifteen years; sought advice for an affection of the right axilla which produced deep and indelible orange-staining of the underwear. There were no subjective symptoms, but the quantity of perspiration in both axillæ was considerable, though not excessive. Upon examination the hair in the right axilla, which was quite abundant, was seen to be dyed a peculiar color and to present a beaded appearance, due to the presence of numerous small brownish nodules encircling the hair-shaft. The most careful examination failed to show any trace of color in the sweat as it was excreted. Some of the hairs were cut off and examined microscopically, with results presently to be described.

Case II.—Mrs. M. R., twenty-nine years of age, brunette, while under treatment for another affection not connected with the skin, called attention to a peculiar staining of the underwear in the axillæ. This staining, while not so marked as in the preceding case, was of the same orange hue and occurred in both axillæ. Upon examination small nodules similar to those already described were found upon the hair, but no coloration of the sweat could be detected, nor was this excretion unusually abundant. There were no subjective symptoms of any kind, treatment being sought only because of the staining of the clothing. It may be of interest to mention the fact that this patient, like the preceding one, was also sterile, although no symptoms of uterine disease were present.

The third case occurred in the person of a male, the disease being limited to the perineal region.

Case III.—F. F., twenty-eight years old, single, of splendid physique, and in excellent health, noticed for several months prior to coming under the writer's observation that the drawers were slightly stained where they came in contact with the perineum. Frequent and careful bathing, with frequent changing of the underwear, failed to prevent the discoloration, and advice was sought as to its causation.

At the first examination nothing abnormal was detected, but the patient was directed to cut off some of the hair in order that it might be examined more thoroughly. Upon examining these hairs with an ordinary pocket-lens, minute brownish nodules were found upon the shaft of the hairs, but the greater number were seated upon the end of the hair-shaft. Upon examination with a magnification of sixty diameters many of the hairs presented a brush-like extremity, in the fibres of which were entangled small nodules. It was evident that the growth of the fungus had caused the hair to break, the distal portion falling off, leaving the nodule imbedded in the brush-like end. Many of the hairs recalled strongly the features presented by trichorrhexis nodosa.

In none of these cases was the sweat colored at the time of its excretion, but it seemed to act as a solvent for the coloring-matter present in the small excrescences upon the axillary and perineal hairs.

A microscopical examination of the nodules with a magnification of two hundred diameters, shows that the fungus began to grow upon the surface of the hair, but with the growth of the parasite the hair itself was penetrated, being split up into numerous small fibres, and, in the case of the perineal hairs, fracture of the hair-shaft occurred with the production of brush-like ends in which the small brown mass was lodged. In the larger nodules a magnification of some eight hundred diameters showed numerous parallel striæ running at right angles to the shaft of the hair, while in the fine colorless hairs upon which the fungus was just beginning to grow, a fine dotted appearance was seen.

Upon subjecting the hair to the action of liquor potassæ and afterward crushing the nodules thoroughly upon a slide, staining with safranin and mounting in glycerin, a high power showed that the mass was composed of numerous micrococci

which grew between the fibres of the hair and around its shaft, and were held together by a yellowish or orange-colored amorphous substance. A few attempts at culture upon sterilized potato at the ordinary house-temperature were made, but without success.

Dr. Kneass, who kindly undertook to make some culture experiments for me in the pathological laboratory of the University of Pennsylvania, succeeded in cultivating the fungus upon agar at a temperature of 37° C. The fungus began to grow within a few hours after implantation, but instead of the brown or orange color seen upon the hair it was sulphur-yellow. Examination of pure cultures showed the cocci frequently arranged in pairs and tetrads, resembling in this respect the micrococcus tetragonus. As the same fungus was obtained from the hair of different patients, it is fair to presume that this micrococcus is the one concerned in the production of the nodules upon the hairs. The fungus grows upon albumin much more slowly.

Although this malady has been known for some time, its true nature has only been determined within a comparatively recent period. Hoffman and Piek in Germany, and Babes in France, first pointed out that the red color was due to a fungus growing upon the hair, which the last-named author succeeded in cultivating upon coagulated albumin. The pigment, according to Babes, who examined it spectroscopically, resembles that produced by the *micrococcus prodigiosus*, the fungus concerned in the production of the so-called "bleeding host." The identity of the two organisms, however, has not yet been proven. By scraping the epidermis in the axillary and perineal regions, organisms similar to those upon the hair have been found, but not all of these were pigmented—many of them were colorless. Balzer and Barthelemy, who have also studied the malady carefully, are of the opinion that it is much more frequent than is commonly supposed, since they were able to collect a considerable number of cases by examining individuals at random. The pigment is not always red, but may be of a yellow hue, and these authors found instances in which the parasite was

present upon the axillary hairs without any pigment accompanying it. In most cases the sweat is unusually abundant, but it is not yet known whether qualitative changes in this excretion are necessary for the production of the disease.

It would seem that blondes are more frequently affected than brunettes, and that weakness and debility are predisposing conditions, although the robust are not exempt.

As has already been mentioned, the sweat is colorless upon its excretion, and for this reason the name chromidrosis should be abandoned; the malady is, in fact, one of the hairs, and not of the sweat-glands.

As the disease is not attended with any annoying symptoms, treatment is rarely sought unless the staining of the underwear is considerable. In the writer's cases alcoholic solutions of bichloride of mercury, two to three grains to the ounce, were employed as lotions with some degree of success; but much more speedy results would probably be obtained by first shaving the affected regions, as in this manner a large part of the fungus would be removed at once.

A STUDY OF THE INFLUENCE OF CHLOROFORM ON THE RESPIRATION AND CIRCULATION:

A Contribution from the Laboratory of Experimental Therapeutics of the Jefferson Medical College of Philadelphia. Being a Report to the Government of His Highness the Nizam of Hyderabad.

By H. A. HARE, M.D.,
PROFESSOR OF THERAPEUTICS IN THE JEFFERSON MEDICAL COLLEGE;
AND
E. Q. THORNTON, M.D.,
DEMONSTRATOR OF THERAPEUTICS, JEFFERSON MEDICAL COLLEGE.

[Read April 5, 1893]

To anyone who endeavors to view the subject of chloroform anæsthesia in an entirely impartial light, two objects raise themselves so high above all others that they form the peaks about which the smaller questions must cluster. The first object on which the judicial eye rests is the firm belief of many clinicians that chloroform may cause sudden cardiac death; the second object to be seen is the statement of the Hyderabad Chloroform Commission that death from chloroform is never due to cardiac failure. In support of the first belief we have not only wide clinical observation, but also the experimental evidence of a number of investigators. In support of the second statement we have an array of experimental study not equalled by any other research extant, associated with an enormous number of negative observations on man. Negative observa-

tions—because Lawrie alone in 25,000 cases has never had a cardiac death.

The controversy concerning the action of chloroform upon the animal organism has been waged so incessantly for many years, and has led to such extraordinary efforts for its elucidation and final decision, that anyone who attempts to take part finds himself almost swamped by the number of statements and opinions which he is forced to regard. We have therefore approached this research feeling it was no ordinary task, and that a path already so well travelled must be gone over with the utmost care if anything new or of value was to be discovered.

In March, 1892, Surgeon Lieutenant-Colonel Lawrie, whose interest in this subject is recognized by the medical profession the world over, wrote to one of us (Hare), asking that another chloroform research be instituted. The express object of the research was the reconciliation of at least some of the contradictory conclusions reached by various experimenters during the past few years.

From the immense number of observations in regard to the action of chloroform in the laboratory and in the operating-room, it is evident that sufficient data are at hand to give us material to reach positive conclusions, and that the contradictory results hitherto obtained must have been reached by misinterpretation and error in experimental method, tinctured perhaps by opinions formed previous to the completion of a line of study. There are certain facts in regard to chloroform which few will deny, the chief of which are that it has the advantages of rapid action without disagreeable preliminary or subsequent symptoms, its bulk is small, and its odor agreeable; but, more important than all, it is much more dangerous than ether.

Though the Hyderabad Commission, in their preliminary conclusions (page 30, paragraph 43), assert that ether is as dangerous as chloroform if given sufficiently to produce true anæsthesia, we believe that the safety of ether is so universally recognized that this conclusion of the Commission can only be excused by the remembrance that ether has probably been used

as little by those who wrote this paragraph as chloroform is used in many parts of America. This possibility is made a probability when we read that "if surgeons choose to be content with a condition of semi-anæsthesia, it can no doubt be produced with perfect safety, though with discomfort to the patient, by ether held rather closely over the mouth. Such a condition of imperfect anæsthesia would never be accepted by any surgeon accustomed to operate under chloroform." That this statement shows, to put it mildly, that the writer knows not whereof he speaks, is proved by the universal employment of ether by hundreds of the best surgeons the world over in preference to chloroform. Further than this medical literature contains so many statistical papers showing the small percentage of deaths from ether as compared with chloroform that this point need not be debated.

There are certain other points in regard to the action of chloroform which may be put aside as settled, and therefore not needing further study, being generally received as beyond criticism, and we have made no experiments looking to their reproduction, but have devoted our efforts solely to the questions over which discussion still proceeds. Thus, all investigators concur in the statement that chloroform, even in ordinary therapeutic quantity, acts as a powerful and constant depressant to arterial pressure. This conclusion has been reached by Bowditch and Minot and Coats, H. C. Wood and H. A. Hare, Gaskell and Shore, the Hyderabad Chloroform Commissions Nos. 1 and 2, and by every experiment in the research now carried out which forms this report. There is no evidence to the contrary, and practically it has never been denied. Gaskell and Shore state, however, that chloroform may cause anæsthesia without lowering blood-pressure, and that chloroform causes primarily a rise of pressure. They also believe that the chief cause of the fall of arterial pressure is cardiac, and not vasomotor depression. The rise we have never seen, except from struggles, and we have never been able to produce anæsthesia without lowering the blood-pressure, even when the drug was used in the smallest quantity capable of causing

anæsthesia and given as slowly as possible. We agree with the statement of the Hyderabad Commission that a fall of blood-pressure always occurs when chloroform anæsthesia is produced.

We are also forced, as the result of our studies, to differ entirely with Gaskell and Shore in their statement that the fall of arterial pressure is due primarily to a weakening of the heart's action, and not to paralysis (depression?) of the vasomotor centre. We believe that both factors cause the fall, but that the dominant factor is vasomotor depression, because, as will be seen in several of our tracings, the pulse waves were quite strong, though the blood-pressure, through vasomotor relaxation, was absolutely down to the abscissa line, and we agree with Lawrie that no conclusions as to the action of chloroform when inhaled can be drawn from the injection experiments of Gaskell and Shore into the arteries. One of our reasons for this belief is the entire difference in method necessary, and the corresponding difference in result. Another reason is that any powerful drug injected into the brain will cause a rise of blood-pressure. Even nitrite of amyl, the prince of vasomotor paralyzants, will do this. (See Gaskell and Shore, p. 17, paragraph 68.) We also believe that results obtained by such interesting methods of experiments as employed by Gaskell and Shore are not capable of giving us positively reliable information, as the conditions are so utterly at variance with those in which chloroform is given to man, and, further, that their methods are such as to give room for fallacious results which cannot be excluded by the greatest caution on the part even of experimenters as experienced as they are.

Secondly, it is not denied by anyone, that we know of, that chloroform exerts a powerful depressant paralyzant action on the respiratory centre. This is agreed to by clinicians and by everyone who has experimentally studied the action of the drug on the lower animals. (See results of collective investigation.) Thirdly, it is universally conceded that chloroform is a lethal agent of great power when brought in direct contact with highly vitalized tissues.

Aside from these facts there are a number of others in which we find ourselves strictly in accord with the conclusions of the Hyderabad Commission. In order to make clear those points in which we agree and differ we print the conclusions reached by the Commission, and to be found on page 17 of their official report :[1]

"1. Chloroform, when given continuously by any means which insure its free dilution with air, causes a gradual fall in the mean blood-pressure, provided the animal's respiration is not impeded in any way, and it continues to breathe quietly without struggling or involuntary holding of the breath, as almost always happens when the chloroform is sufficiently diluted." "As this fall continues the animal first becomes insensible, then the respiration gradually ceases, and, lastly, the heart stops beating."

In every one of our experiments this course was followed as soon as struggles ceased. (Hare and Thornton.)

"If the chloroform is less diluted the fall is more rapid, but is always gradual so long as the other conditions are maintained; and however concentrated the chloroform may be, it never causes sudden death from stoppage of the heart. The greater the degree of dilution the less rapid is the fall, until a degree of dilution is reached which no longer appreciably lowers the blood-pressure or produces anæsthesia."

With this entire statement our results are practically in accord, but we would like to qualify the words "however concentrated chloroform may be, it never causes sudden death from stoppage of the heart," by saying it never has caused sudden stoppage of the heart in any of our experiments unless respiration ceased primarily. We make this modification because, as we will point out later in this report, we believe circumstances may exist in which the diseased heart may stop

[1] These are not the final conclusions, published in the Lancet and elsewhere, which were drawn up for general readers, but from the more scientific and accurate deductions of the research itself.

suddenly under chloroform. On the other hand, we do not believe that it is possible in lower animals (the dog) to cause cardiac death by the freest possible use of chloroform by inhalation without causing primary respiratory arrest, and respiratory arrest having taken place the death which follows is partly due to asphyxia and partly to direct cardiac failure and vasomotor paralysis.

In reaching this conclusion one of us (Hare) is well aware that his position is directly opposed to his conclusions published in a joint paper by H. C. Wood and himself in the *Medical News*, of February 22, 1890. The tracings shown in that paper to prove that chloroform was capable of causing primary arrest of the heart are all (except No. 5) tracings of experiments in which chloroform was *injected directly into the jugular vein*, which is an entirely different thing from its absorption from the lungs into the blood well mixed with oxygen. Even quinine injected into the jugular vein will cause cardiac arrest. Further than this, this research has shown us that in only one of the three tracings presented by Wood and Hare did death *certainly* occur from cardiac failure, even when the drug was given intravenously, namely, in their tracing No. 4. Perhaps if artificial respiration had been resorted to recovery would have occurred, as our experiments have proved is possible even after both heart and respiration have apparently ceased. The pulse line in tracing No. 3 (Wood and Hare) is more than reproduced by jugular injection in No. 14 in this research, and No. 5 (Wood and Hare), by inhalation in No. 23 of this research, and it will be noted that though the heart failed in these experiments to make a pulse mark, for the time being, yet it eventually recovered sufficiently to do so or was found beating when the chest wall was opened or a needle thrust into the heart. Again, in experiment No. 13 in this research, it will be seen that when the pen was practically at the abscissa it suddenly rose again. The heart was in all these experiments very weak, but it recovered rapidly from its weakness, so we must conclude that the cardiac condition was one of weakness or depression, and not paralysis or death.

"2. If the inhalation is interrupted at any stage, the fall of pressure still continues at a rate which depends altogether on the rapidity of the fall while the chloroform was being inhaled. This after-fall is probably due to absorption of a portion of the residue of chloroform in the air-passages after the stoppage of the inhalation. In this way it often happens, if chloroform is given rather freely, that though the respiration may be going on when the chloroform is discontinued, it afterward stops."

Our results are identical with this conclusion.

"3. If the administration of the chloroform is stopped at an early stage the pressure very soon begins to rise again, and gradually becomes normal." [See our experiments Nos. 1, 13, 17, 23, and 27 (Hare and Thornton).] "But if the chloroform is pushed further, there comes a time, not easy to define, when the blood-pressure and respiration will no longer be restored spontaneously, although the heart continues to beat after the inhalation is stopped."

See our experiments Nos. 1, 10, 13, 14, 16, 17, 18, 23, and 27, all of which confirm this conclusion (Hare and Thornton).

"4. If the fall has been very gradual it may occasionally happen that the respiration stops completely, and still the blood-pressure rises again, the respiration recommencing spontaneously in the course of the rise." [See our experiments Nos. 12, 13, 17, 23, and 26 (Hare and Thornton).] "In the same way when the inhalation has been discontinued, the respiration may stop during the after-fall of the blood-pressure and begin again spontaneously. As a rule, if the respiration has stopped, or even becomes slow and feeble at the time when the inhalation is discontinued, and artificial respiration is not resorted to, the fall in blood-pressure will continue until death ensues."

See end of Nos. 1, 10, 13, 17, 18 (Hare and Thornton).

"5. There are two conditions which frequently disturb the gradual fall of the blood-pressure, viz., struggling and holding

the breath, and it is only by great care that they can be avoided in animals."

Our results are entirely in accord with this.

"6. Struggling, independently of any change in the respiratory rhythm, appears generally to raise the blood-pressure. In one case of a dog much weakened from phosphorus the pressure fell every time he struggled."

"7. When struggling is accompanied, as it often is, by acceleration of the respiration and pulse, especially if the respiration is deep and gasping, it leads to a more rapid inhalation of chloroform, and consequently to a more rapid fall of blood-pressure and a greater after-fall. In order to keep the chloroform cap or inhaler in its place during the animal's struggles, the administrator is obliged to hold it down more tightly over the nose and mouth, and this materially assists in hastening the rapidity of the inhalation, and consequently of the fall in blood-pressure."

We entirely concur in this.

"8. The effect of involuntarily holding the breath, which, as anybody can prove by experiment upon himself, must happen when an inhaler saturated with chloroform is first applied to the face, is much more remarkable, the pressure often falling with great suddenness while the heart's action is markedly slowed. As soon as the animal draws breath again the pressure rises as suddenly as it fell, but the gasping inspiration which succeeds then causes very rapid inhalation of chloroform with immediate insensibility and a rapid fall of blood-pressure which quickly becomes dangerous."

See experiments Nos. 1 and 5 of Wood and Hare. See experiments Nos. 13 (parts 3, 5, 6) and 17 and 27 (Hare and Thornton). This fall is sometimes dangerous and sometimes not; certainly not a dangerous fall in tracing No. 16 under atropine. As a whole, we concur in this, and believe that the first action is solely due to reflex inhibition through vagal and trigeminal irritation (Hare and Thornton).

"9. The combination of struggling with alternate holding the breath and gasping, which results if chloroform is applied closely to the face without sufficient dilution with air, causes violent fluctuations, and then a speedy fall of the blood-pressure, which very soon leads to a dangerous depression with deep insensibility and early stoppage of the respiration. The after-fall, under these circumstances, is rapid and prolonged. It is this combination of events which causes struggling animals to go under chloroform so quickly."

See 13, 16, 27 (Hare and Thornton). We also find it rapid.

"10. The effect of holding the breath may occasionally cause a temporary fall of blood-pressure after the chloroform inhalation has been stopped, or even when the animal is quite out of chloroform. This fall is recovered from directly the animal breathes again."

We concur in this.

"11. Slight continuous asphyxia, such as is produced by pressure on the neck by straps, a badly fitting muzzle, or hindrance of the chest movements by the legs being too tightly bound down, gives rise to exaggerated and irregular oscillations of the blood-pressure, and slowing and irregularity of the heart's action. If it leads to, or is accompanied by, deep gasping inspiration, it is apt, like anything else which causes this, to increase the intake of chloroform and bring about a rapid decline of blood-pressure."

We concur in this conclusion.

"12. Complete, or almost complete, asphyxia, as by forcibly closing the nose and mouth, or closing the tracheal tube after tracheotomy, has an effect similar to, but more marked than, that produced by holding the breath, and the character of the trace corresponds precisely to that produced by irritation of the peripheral end of the cut vagus. The pressure falls extremely rapidly, sometimes almost to zero, and the heart's action becomes excessively slow or even stops for a few seconds."

"13. This effect of asphyxia is the result of stimulation of the vagi. The proof of this is (*a*) that the trace corresponds exactly, as stated above, to that produced by direct irritation of the vagus; (*b*) division of both vagi entirely abolishes it; and (*c*) the administration of atropine, which paralyzes the vagus, also abolishes it."

See tracing 15 (Hare and Thornton). That vagal irritation does account for some of the circulatory disturbance is no doubt true, but in reality the changes are chiefly vasomotor in character. We have not found that either vagal section or vagal paralysis with atropine prevented these phenomena, although it may modify them. See tracings Nos. 10 and 17 (Hare and Thornton).

"14. In trace 158 (Fick 4), which was taken during asphyxia after a full dose of atropine, it will be seen that there is an alternately slow and rapid pulse, according to the phase of the respiratory movement; but no continued slowing of the heart, as in vagus irritation. But there was still a distinct fall of pressure after the atropine when the breath was held, and it was thought that the slowing of the pulse above noted in this condition might be due to the disturbance of the heart from tension in the pulmonary vessels in the absence of respiratory movements, rather than to irritation of the vagi. To test this point experiment No. 184 was instituted. In this experiment the dog's chest was forcibly inflated with bellows connected by a tube with the trachea, and the effect of this proceeding was to cause a fall of pressure and slowing of the heart exactly the same as in involuntary holding of the breath. The dog was then poisoned with atropine, after which inflation of the chest still caused a fall of pressure, but without slowing of the heart (*vide* Fick Nos. 8 and 9). The fall of pressure must be in some degree independent of vagus irritation, which, however, usually accompanies it."

"15. It only remains to be considered whether the slow action or temporary stoppage of the heart, with great fall of pressure produced by vagus irritation, is in itself an element of

danger in chloroform administration, and if it is not, wherein the danger actually lies."

See note to paragraph 16. (Hare and Thornton.)

"16. The experiments in which deliberate irritation of the vagi was carried on during anæsthesia show unmistakably that irritation of these nerves diminishes rather than enhances the danger of anæsthetics. The effect upon the heart is never continuous, and as the vagus becomes exhausted, or when the irritation is taken off, the blood-pressure rises again, as it does when the same result is produced by asphyxia. The slowing of the heart and circulation which is produced by irritation of the vagus by any cause, such as holding the breath in chloroform administration, retards the absorption and conveyance of chloroform to the nerve centres, just as holding the breath, whether voluntary or involuntary, prevents chloroform from entering the lungs; and of itself slowing or temporary stoppage of the heart in chloroform administration is not dangerous."

We shall point out later in this report how we differ from this conclusion, for we believe that true fatty heart, plus ventricular engorgement, plus vagal irritation, plus possible valvular disease, and finally plus extreme vasomotor relaxation, may result in death in frightened persons. While this is not, scientifically speaking, a cardiac death from chloroform, practically the chloroform is the last straw which upsets the cardiac balance. However, Lawrie and his colleagues recognize this as well as ourselves. (See paragraph No. 38 of the Hyderabad Report.)

"17. To answer the second part of the last question in paragraph 15 is easy enough, if it is kept in mind that the effect of vagus irritation upon the heart is never continuous; and in chloroform administration, as the pressure rises again after the slowing of the heart and temporary fall of pressure produced by any form of asphyxia, violent respiratory efforts with bounding heart's action lead, as in the case of struggling, to a rapid and dangerous inhalation of chloroform, and consequent rapid and dangerous decline in blood-pressure. It is, in fact, the tem-

porary exhaustion of the vagi after stimulation that is to be feared, and not the actual stimulation as long as it is continued."

We believe that in the *healthy* animal or man this is true.

"18. In accordance with this fact it will be found that in chloroform administration neither holding the breath, even if involuntary, or vagus inhibition, can be kept up beyond a certain time; and if the chloroform is not removed from the face, one or both of two things may happen: (1) when the animal breathes again it takes deep and gasping inspirations, the lungs become filled with chloroform, and an overdose is taken in with extreme rapidity; or (2) when the restraining influence of the vagus is taken off the heart, through the irritation ceasing or the nerve becoming exhausted, the heart bounds on again, and the circulation is accelerated in proportion. The blood then becomes quickly saturated with chloroform, and an overdose is at once conveyed to the nerve centres. The theory which has hitherto been accepted is that the danger in chloroform administration consists in the slowing or stoppage of the heart by vagus inhibition. This is now shown to be absolutely incorrect. There is no doubt whatever that the controlling influence of the vagus on the heart is a safeguard, and that it is the exhaustion of the nerve which is dangerous."

We believe this is true of the healthy heart, but not of one engorged with blood, particularly if it has undergone fatty degeneration. This is only a hypothesis, however, as we have no experimental basis for this belief.

"19. It can be readily understood how a condition in which the pulse is rapid and bounding with high blood-pressure leads to more rapid absortion of chloroform from the lungs, and a more rapid propulsion of the chloroformed blood to the medulla oblongata, and consequently to a more rapid paralysis of the respiratory and vasomotor centres and precipitous fall in the blood-pressure. Such a condition is produced in some cases by ether, or by division of both vagi or by a full dose of atropine. Not only is the poisoned blood carried more swiftly to the vital centres in these cases, but added to this there is the

fact that as the heart is already doing its utmost before the chloroform is given, it is unable to stave off, by increased work, the fall in pressure that occurs when the vasomotor centre is paralyzed. On the other hand, it seems clear from experiment No. 92 that the direct action of chloroform upon the heart's substance is not the cause of the fall of pressure that occurs when it is inhaled."

That we (Hare and Thornton) agree with these conclusions (paragraphs 15, 16, 17, 18, and 19) is shown by the following extract from the research of Wood and Hare, in the *Medical News*, February 22, 1890: "The theory has from time to time found advocates that the vapors of chloroform may, by irritating the larynx and adjacent parts, cause arrest of the heart through a reflex inhibition. To test the possibility of this we have made a number of experiments. When the tracheal canula is tied tightly into the trachea some distance below the larynx, it is evident that the latter organ is isolated from the general respiratory tract, and that chloroform injected into it will exert only a local influence. In all the experiments which we have made in the way just indicated, the injection has been followed by an immediate and very pronounced primary fall of the pressure, followed, after a very brief interval, by a rise which usually reaches decidedly above the norm. See tracing (Wood and Hare). The primary fall of arterial pressure, which has just been spoken of, can scarcely be produced except by reflex inhibition of the heart or of the vasomotor centres, whilst the secondary rise is probably the result of reflex vasomotor spasm. In order to throw light upon this question, we have made experiments by injecting chloroform into the larynx after division of the pneumogastric nerves, the trachea being ligated so as to prevent the entrance of the anæsthetic into the lungs." Tracing 2 (Wood and Hare).

In making practical application of the experiments just discussed, it must be noted that in no case have we succeeded in completely arresting the heart's action by injecting chloroform into the larynx, and as the chloroform was injected in liquid form, it is plain that the irritation was more intense than could

be produced by the mere vapors of the anæsthetic, however concentrated; therefore, while it must be considered that it is possible for a reflex inhibitory arrest of the heart to occur during the inhalation, such an accident is extremely improbable, and we consider it practically certain that a heart so arrested could, a few seconds later escaping from the inhibitory control, recommence its beat. See No. 15 (Hare and Thornton). It certainly has never been proved that chloroform can cause in the human subject permanent reflex inhibitory cardiac arrest, and as our experiments upon the dog have failed to cause such arrest, we consider it very improbable that cardiac arrest is ever produced in man by chloroform. To this we wish to add: unless cardiac disease or condition is such as to be unable to withstand any shock whatever (Hare and Thornton).

"20. In experiment No. 92 repeated injections of 20 m. of chloroform were made into the jugular vein, and its effect was not to paralyze the heart, but to produce anæsthesia and a gradual fall of blood-pressure exactly as if the chloroform had been inhaled. In experiment No. 72, after a considerable amount of ether had been injected into the jugular vein and a bounding condition of pulse had been produced, the effect of injecting chloroform into the jugulars was much greater, and the fall of blood-pressure much more rapid and dangerous than in the case when chloroform alone was injected. Granting, then, the truth of Ringer's conclusions from experiments on the frog's heart (which have not been repeated and confirmed by the Commission) that chloroform has a gradual paralyzing effect upon the heart's tissue, we must conclude that such an effect, in the degree in which alone it could occur in the practical inhalation of chloroform, would rather be a source of safety than of danger."

With these conclusions we have to entirely disagree, and we cannot understand how results so completely at variance with our own and with those of Wood and Hare could have been arrived at. (See tracings Nos. 19 and 20.) The tracings we obtained do not admit of wrong interpretation. Perhaps the

difference lies in the fact that in our studies enough water was added to the chloroform to carry it *en masse* to the heart, whereas in the East Indian studies the pure undiluted drug was injected and had not sufficient volume to reach the heart, and was gradually volatilized in the vein, and so produced a gradual effect. This is shown in tracing No. 8 (Hare and Thornton). We find the injection of from 2 to 4 c.c. of chloroform into the jugular vein causes arrest of respiration, rapidly followed by cardiac arrest, which is not due to respiratory failure.

It having been denied that chloroform, when injected into the jugular vein, causes cardiac depression, and the recent experiments of Leaf and his colleagues in Hyderabad having been cited to support this view, let us discuss the facts before us. In these researches they found that when chloroform was given in excessive amount, the pen fell to the abscissa line and failed to record a pulse-wave, whilst a needle in the heart muscle continued to beat for many seconds. While at first glance these results, which we have also obtained (see Nos. 19 and 20) seem to prove that the cardiac arrest is only apparent and not real, in reality they have been given an importance far in excess of their value. We have proved again and again that the movement of the needle may be due not to a true cardiac contraction, but to incoördinated contraction of one of the ventricles, and to contractile movements of bands of the cardiac muscle, which movements are very often rhythmical enough to make the needle beat regularly. We have found this needle movement taking place even after an injection of chloroform had been sent directly into the heart through the chest wall, and in a heart the muscle of which failed to respond to any strength of faradic irritation, except in those parts which had not come in contact with the poison.

Further than this, if an animal be given very large doses of chloroform intravenously, or by direct intra-cardiac injection, respiration invariably ceases if the amount be large enough; and if the chest be opened, one condition will be constantly found, viz., the heart so widely dilated as to fill the pericardium almost to bursting, and the cavities, particularly the

ventricles, engorged with blood. Although they may still be feebly contracting, the contraction is abortive, and fails to cause arterial flow. If the drug has been used intravenously, by the jugular, the blood in the right and left heart will be found red provided death has come on rapidly. If not it will be dark and venous. The lungs will be found of the peculiar pink hue due to altered blood. If the injection has been cardiac it will be found that the ventricle with which the poison has come in direct contact has become paralyzed, while the other is making fairly good efforts to work. (See No. 21, Hare and Thornton.) When the injection takes place into the right ventricle, so that the poison passes to the left cavity, both ventricles fail to act voluntarily or to faradism. (See experiment No. 25, Hare and Thornton.) Chloroform is capable, therefore, of causing death of the cardiac muscle whenever it comes in contact with it, and that there is no possibility of this arrest being due to vagal irritation is proved by experiments in which vagal section preceded the use of the chloroform. (See Nos. 22, 24, 25.) We doubt if the last sentence of paragraph 20 is justified by our present knowledge.

"21. The Committee discussed the advisability of cutting the vagi some time previous to experimenting on the blood-pressure with chloroform. The effect of this procedure is to cause continuous rapid action and tendency to exhaustion of the heart, as well as to degeneration of the terminal branches of the nerves in the heart if the animal lives sufficiently long. Such experiments might be of some interest theoretically, and also have had a practical bearing upon the condition of the heart in certain cases of chronic alcoholism; but the Committee decided not to perform them, as it considered the end to be gained did not justify the pain they would have inflicted."

We have not made this particular experiment, but we found that vagal section immediately before the drug was used did not materially alter the result.

"22. In experiment No. 178, the case of a dog that had had morphine, remarkable slowing, and even temporary cessation

of the heart's action occurred again and again at the same moment as the respiration stopped, but the heart invariably recovered itself, and began again to beat regularly before any steps were taken to restore the animal, and without any respiration occurring. We find in this case that it was possible to restore the animal even after unusually long intervals had been allowed to elapse between the cessation of natural and the commencement of artificial respiration. The failure of the heart, if such it can be called, instead of being a danger to the animal, proved to be a positive safeguard by preventing the absorption of the residual chloroform and its distribution through the system."

We have not made any experiments on this point, but it seems to us that the known facts quite justify the conclusions.

"23. The effect of artificial respiration after the natural respiration has ceased, is to cause an alternate rise and fall of small amount in the blood-pressure, the trace thus formed upon the drum being a coarse imitation, altered somewhat by the shaking of the table, of the natural respiratory curve. The difference consists chiefly in the fact that the artificial rise and fall is more abrupt than in natural breathing, and that the rise always coincides with expiration or compression of the chest. After artificial respiration has been continued for a certain time the blood-pressure begins to rise again, and a little later natural respiration returns."

See experiments Nos. 3 and 27 (Hare and Thornton.) We concur in this.

"24. The effect of artificial respiration in restoring an animal after the respiration had stopped was always marked. In a few exceptional cases, such as No. 159, a phosphorus dog, and No. 142, a horse which had an enormous overdose, although the artificial respiration was commenced as soon as possible after the breathing was noticed to have stopped, it was not successful."

See our experiments Nos. 3 and 27 (Hare and Thornton). We concur as qualified by the next paragraph.

"25. Complete stoppage of the respiration always means that an overdose has been administered, and the overdose may have been so great as to render restoration impossible. It is impossible to say whether, after chloroform has been pushed and then discontinued, the respiration will be restored spontaneously or not, and it is never in any case certain that artificial respiration will restore the natural respiration and blood-pressure, no matter how soon it is commenced after the respiration stops. A great deal depends upon the amount of the after-fall; in some cases, even after the respiration has been restored, the pressure continues to fall and respiration again ceases, and artificial respiration then fails. We thus find respiration restored by artificial respiration while chloroform is still being absorbed, and this tends to show that artificial respiration does not merely pump the chloroform out of the blood, but exerts considerable influence in exciting the natural respiration."

See our experiments Nos. 1, 3, 5 (Hare and Thornton).

"26. The time which elapses before artificial respiration succeeds in restoring natural respiration varies very greatly. In one case, No. 116, it was continued for eleven minutes before the first natural gasps commenced. This period is undoubtedly prolonged in some cases by a condition of physiological apnœa which renders it unnecessary for the animal to breathe. Consequently, whenever the pressure rose considerably during artificial respiration it was stopped, and the animal then generally breathed a few seconds."

Whenever apnœa developed the fall of pressure would persist, and a rise not take place.

"27. The time which may be allowed to pass with impunity before commencing artificial respiration also seems to vary considerably. This point was not particularly attended to in the manometer experiments; except in experiments 162 and 178, which were instituted to test the truth of the opinion formed by the Sub-Committee, that morphine had some slight action in impairing the efficiency of artificial respiration. In

these cases the commencement of artificial respiration was postponed for more than two minutes after respiration ceased, and was successful; but this is certainly far above the average interval that can be allowed with safety. The success of artificial respiration in restoring the blood-pressure is in some cases very remarkable. In experiment No. 40 the heart had apparently ceased beating, and the dog was believed by everyone present to be dead, and yet recovered with artificial respiration. The success in this instance is due to the fact that concentrated chloroform had been pushed for two minutes, regardless of the breathing, and the stoppage of the heart was due to stimulation of the vagus through asphyxia. The animal was, therefore, easily restored, as he was suffering more from asphyxia than from chloroform poisoning."

"28. It corresponds to those cases, which are so often reported, in which dangerous failure of the heart is said to have occurred some minutes after the administration of chloroform had been discontinued, and which are sometimes restored, and sometimes not, by artificial respiration. There is nothing at all sudden about the failure of the heart in these cases, but the attention of the chloroformist, which has been wandering, is suddenly called to the fact that the patient is apparently dead. When the animal was really dead, it was found in some cases that artificial respiration still maintained a small amount of mean pressure in the manometer. In others the pressure seemed to fall to the zero line between each compression of the chest."

We reach a similar conclusion.

"29. The dangers of too vigorous artificial respiration were illustrated in some of the accidental deaths. In one case the liver was badly ruptured, and in another the pleural cavity was full of blood. In three cases, Nos. 80, 92, and 103, rhythmical movements of the diaphragm were noticed after the heart had ceased beating, and after the chest had been opened. It is remarkable that in two of these cases the splanchnic nerve had been divided. The third was a case in which chloroform had

been injected into the jugular, and in this case there was a synchronous movement of the jaw as well. In all, death and stoppage of the heart had occurred gradually, and in No. 103 the heart was still irritable. These movements cannot be called respiration, though the last gasp of a dying animal, that ineffective jerk of the diaphragm, which is such a fatal symptom, is very likely in many cases a movement of the same character. Similar movements, which were continued much longer, occurred in experiment No. 104 after the thorax was opened, while the heart was still beating. Still more remarkable convulsions of the muscles of the jaws, ears, and fore-feet occurred in experiment No. 167, in the case of a dog that had been poisoned with nicotine. These movements continued at regular intervals for more than ten minutes after death, and were sufficiently forcible to jerk the handles of a pressure-forceps fixed on the end of the tongue off the table at each spasm. In a rabbit, in experiment No. 153, the auricles of the heart continued to beat rhythmically for three hours after it was supposed to be dead from chloroform and its thorax had been laid open. Irritability of the heart after death was noticed in many cases, but seemed to be most marked in cases where ether had been used."

We have made no studies in regard to this point.

"30. Chloroform injected in the heart through the jugular vein did not cause clotting of the blood, as was the case when ether was injected."

Chloroform did cause clotting in our experiments.

"31. In the course of the experiments of the Committee various drugs were administered in order to ascertain if they had any effect in modifying the action of chloroform. The result showed that none of them had any effect in preventing the typical descent of the blood-pressure that occurs when chloroform is inhaled. Atropine, when given in a dose sufficient to paralyze the vagi, of course, prevents the action of those nerves in asphyxia, and by increasing the action of the heart it appears

to cause a more rapid descent in the blood-pressure when chloroform is inhaled, as has been already explained."

We cannot agree to this. See experiments 13, 16, 17, and 18 (Hare and Thornton).

"Morphine appeared, in No. 162, to render the rise in blood-pressure that occurred when the chloroform was discontinued slower and less complete, and to bring about a more or less permanent condition of anæsthesia. It may be noted that the animal used in this experiment was a monkey, and in other experiments with monkeys, when no morphine had been given, it was remarked that the animal, after a few inhalations of chloroform, would often lie quite quiet in a state of semi-insensibility for a long time without further inhalations; still this condition was much more marked in experiment No. 162 than in any of the others. No action of this kind was noticed in the dog, No. 178, but other experiments (Nos. 90 and 94) showed that pariah dogs are very indifferent to the action of morphine, and it is probable that the dose of morphine in this case was insufficient to bring about the condition noted in the monkey. The peculiar behavior of the heart in No. 178 was not the result of the previous administration of morphine, for a similar phenomenon had occurred in other cases (49 and 60) in which no morphine had been given. Experiments Nos. 162 and 178 prove conclusively that morphine has no effect in shortening the period that may be allowed to elapse between the cessation of natural respiration and the commencement of artificial respiration."

We have already shown, in Nos. 10, 16, 17, and 18 of our experiments, that atropine seems to produce a very gradual fall of pressure, and to preserve the circulation. With the other drugs we have not experimented.

"32. The other drugs used had no effect upon the action of chloroform except when their own special action became the leading feature in the case, as, for instance, during the vomiting from apomorphine (104, Fiek 9) or the convulsions produced by nicotine (167)."

"33. In order to test the alleged danger from shock during chloroform administration, the Commission performed a very large number of those operations which are reputed to be particularly dangerous in this connection, such as extraction of teeth, evulsion of nails, section of the muscles of the eye, snipping of the skin of the anus, etc. In many cases the operation was performed when the animal was merely stupefied by the chloroform, and not fully anæsthetized. In such cases a slight variation in the blood-pressure would sometimes occur, such as one would expect from the irritation of a sensory nerve or from the struggling that ensued, but in no case in any stage of anæsthesia was there anything even suggestive of syncope or failure of the heart's action. In thrusting a needle into the heart, there was often a momentary, but well marked, fall of blood-pressure; but even this was absent in all other injuries. If chloroform really had any power to increase the tendency to shock in operations, it is impossible to believe that it would not have been manifested to some degree at least in one or other of these numerous experiments. The Commission was, however, not content with this negative result, and determined to ascertain the effect of direct irritation of the vagi during continued chloroform administration. The result of such experiments (Nos. 65, 117, and others) proved that inhibition of the heart's action prevented, rather than assisted, the fatal effects of prolonged chloroform inhalation. An animal that was put into a condition of extreme danger (from which it could only be restored by means of artificial respiration) by inhalation of chloroform for one minute, recovered spontaneously and readily after five minutes of chloroform inhalation together with inhibition of the heart by electrical irritation of the vagus carried on simultaneously. In one of these experiments, No. 117, chloroform was pushed for seven minutes, and during continued irritation of the vagus the animals repeatedly came round without artificial respiration. The danger really begins when the irritation is discontinued or fails to inhibit the heart, and thus enables the chloroform in the lungs to be rapidly absorbed and thrown into the system. This danger is certainly

increased by deliberately pumping the chloroform into the lungs by means of artificial respiration, for animals in which this was done, although they showed a tendency to recover when the chloroform and irritation of the vagus were discontinued, afterward died rapidly."

"34. On another occasion, during experiment No. 117, the animal was very nearly killed by a comparatively short inhalation of chloroform, owing to the electrodes becoming accidentally short-circuited and failing to keep up the irritation of the vagus. Something similar occurred in No. 177, the effects of irritation of the vagus passing off, while the chloroform was still being pushed, and thus putting the animal into a condition of extreme and unexpected jeopardy. Nothing could be more striking than these near approaches to accidental death from failure to irritate the vagus efficiently."

"35. Other experiments were made to test the truth of the statement that chloroform increased the action of electrical stimuli applied to the vagus, and showed conclusively that it has no such effect. In one instance only the inhibition seemed to be intensified as the chloroform was commenced, and diminished when it was discontinued, but apart from the fact that the supposed effect ceased much too suddenly, a repetition of the experiment on the same and other animals showed that there was in reality no such effect. The increased inhibition in this instance was due to the chloroformist compelling the attendant who was holding the electrodes to change his position, and thus making him unconsciously apply them more efficiently. When the chloroformist withdrew they were restored to their former position. This affords an instance of the care that has to be taken in making experiments, if one is not to be deceived."

"36. To test the effect of shock due to vasomotor change rather than affection of the heart, Goltz's experiment on the frog was repeated on three dogs. In one there was slight lowering of the pressure, which was not extensive, but in the others no effect was produced at all. Other operations which seemed likely to produce shock, such as violent blows upon the testicle, were singularly devoid of effect. Failing to lower the

blood-pressure by any of these methods, recourse was had to section of the splanchnics, but the low condition of blood-pressure this produced appeared, like stoppage of the heart from vagus irritation, to be a source of safety rather than of danger during chloroform administration. In this connection experiment No. 111 may be studied. There was not much external hemorrhage, but the splanchnics were divided—a proceeding which, as is often said, bleeds the animal into his own vessels. The pressure was after this extremely low, but chloroform was repeatedly given, and various other actions taken, and then chloroform had to be pushed on a saturated sponge enclosed in a cap for eleven minutes before respiration ceased."

"37. The experiments on dogs that had been dosed with phosphorus for a few days previously show that the fatty, and consequently feeble, condition of the heart and other organs so produced has no effect in modifying the action of chloroform. The ease with which vagus irritation and the Glasgow trace could be produced in these animals, by even slight degrees of asphyxia (*vide* 148), was very remarkable, but this was equally the case in dogs that had been given phosphorus only a few hours before the experiment, and whose organs were not yet fatty (*vide* 156). Many of these cases were in the last stage of phosphorus poisoning, and several of their companions died without any experiment having been performed on them before or on the same day as they died (*vide* the low state of blood-pressure in No. 163). Numerous attempts were made in these animals to produce shock by operations in the recumbent and vertical positions, but without any more result than in those that were healthy."

We have no experience to offer.

"38. The truth about the fatty heart appears to be that chloroform *per se* in no way endangers such a heart, but, on the contrary, by lowering the blood-pressure, lessens the work that the heart has to perform, which is a positive advantage. But the mere inhalation of chloroform is only a part of the process of the administration in practice. A patient with an extremely fatty heart may die from the mere exertion of getting upon the

operating-table, just as he may die in mounting the steps in front of his own hall door, or from fright at the mere idea of having chloroform or of undergoing an operation, or during his involuntary struggles. Such patients must inevitably die occasionally during chloroform administration, and would do so even were attar-of-roses or any other harmless vapor substituted for chloroform."

We agree entirely with this statement, but as chloroform has confessedly some cardiac action, and a very positive vasomotor and respiratory effect, the fatal result must be more direct.

SUMMARY.

Having given what evidence we have accumulated, let us see what practical deductions may be drawn.

From a careful study of the experiments so far reported, from studies made by one of us some two years ago with H. C. Wood, and finally from our own series of experiments, we believe that the question can be settled by the acceptance of both views in a modified form.

We very positively assert that chloroform practically always kills by failure of respiration when administered by inhalation, provided, and this provision is most important, that the heart of the anæsthetized is healthy and has not been rendered functionally incompetent by fright or violent struggles, or, again, by marked asphyxia. By a healthy heart we mean one which has not undergone true fatty degeneration, or has not so severe a valvular lesion as to make the slightest variation in the even tenor of the circulation fatal.

As positively as we assert that chloroform kills primarily by respiratory failure, so do we also assert that in excessive dose by inhalation it has a depressant effect on the circulation, which is chiefly due to centric vasomotor depression, with final depression of the cardiac muscle itself. Depression of the cardiac muscle alone is never great enough to cause death when the chloroform is given by inhalation, but we believe that gradual asphyxia with the direct depression of the circulation may do

much toward producing a fatal result, for vasomotor integrity is almost as necessary to life as an intact cardiac mechanism. The circulatory failure produced by chloroform has been considered a safeguard because it was supposed to prevent chloroform going to the vital centres, but in reality it is no "safeguard," because profound circulatory depression is as great an evil as respiratory narcosis. That the circulatory depression may be dangerous is not only apparent, but it is stated to be so by the Hyderabad Commission at the end of paragraph 8. This circulatory depression may be so profound that recovery is impossible, even with the most thorough artificial respiration, a fact stated by the second Hyderabad Commission in paragraph 25, which we quote in this paper. This emphasizes the fact that we cannot afford to totally ignore the effect of chloroform on the circulation, and we cannot consider the patient in danger of circulatory failure only when the respiration *ceases*, but as soon as it become abnormal. On the other hand we should remember that even if chloroform has been given properly. the arterial pressure may be so low as to give no pulse in the radial artery, and yet the circulatory system may be so restored at once when the drug is removed. If, therefore, the chloroform is properly administered, is there danger of the circulatory effect in man? We think that it is just at this point that our research, and every other on animals, fails, and necessarily fails, to produce a positive reply. The variation in the action of a drug on a diseased individual from its effect on the normal one is notorious, and we have no right to dogmatically assert that there is absolutely no danger of circulatory depression in man even if we found no evidence of failure in dogs, because there may be many idiosyncrasies or variations through disease in the human being which may completely reverse the results of experiments made upon healthy animals.

In other words, supposing that the amount of depression from very full doses of chloroform equals 25 units; this amounts to little in the normal heart, but if the heart be depressed 25 additional units by disease, the depression of 50 units may be

fatal, particularly if to this 50 is added 25 units more of depression through fright and cardiac engorgement, through disordered respiration or struggling. That true depression of the heart muscle may take place under chloroform seems to us most undoubted, and we think that the tracings in every research that we have seen support this view. There is always a decrease in cardiac power manifested by the decrease in the force of the individual pulse beat, and this passes away only if the chloroform is removed early enough. We also agree with McWilliam, that from the very first inhalation of chloroform there is a constant tendency to cardiac dilatation.

We come, finally, to the all-important questions:

First. Is chloroform a safe anæsthetic?

Second. Are we to watch the pulse or respiration during the use of the drug, and what are the signs in the respiratory function indicative of danger to the patient?

Third. What is the true cause of death from chloroform?

Fourth. Is death from chloroform possible when it is properly administered?

Fifth. Under what circumstances is the surgeon to use chloroform in preference to the less dangerous anæsthetic ether?

Sixth. What is the best way of administering chloroform?

1. To the first question the answer is, Yes, for the majority of cases, provided it is given by one who is skilled in its use, and not only knows how to give it but to detect signs of danger. It is not as safe as ether at any time, other things being equal, and never so safe in the hands of a tyro.

2. To the second question the answer is, Watch the respiration, because, as soon as enough chloroform is used to endanger the circulation the respiration will show some signs of abnormality either in depth, shallowness, or irregularity. In other words, the very effect of the drug may be to cause such deep and rapid respirations that an excessive quantity of the drug is taken into the lungs and continues to be absorbed even after the inhaler is withdrawn.

As there is always a fall in pressure under chloroform it is

difficult to feel the radial or temporal pulse, and the respiration centre recognizes the degree of arterial depression which its sister vasomotor centre has permitted, by finding that its blood supply is insufficient. As respiration fails first it should be watched first. Finally, it is only by watching the respiration that we can tell how much chloroform the patient is getting. We do not watch this function for danger alone, but to tell us of the dose.

3. The answer to question 3 is that death is always due in the healthy animal to respiratory failure, accompanied by circulatory depression, which latter may be severe enough to cause death, even if artificial respiration is used skilfully. Death only occurs in the most healthy animal when chloroform is given in excessive quantities.

4. Question 4 is impossible to answer for man from the basis of experimentation, as we cannot produce identical diseased states in animals with those developed under various conditions in man. The physician having a case of heart disease should always advise the patient of the danger of any anæsthetic, and he should remember, whether it is wise to tell the patient or not, that anæsthesia always means a step toward death in the healthiest of men. In the event of a death under chloroform the physician is not to blame if he has taken proper preliminary precautions, and given the chloroform properly.

Everyone is agreed that the patient taking chloroform should have plenty of fresh air, and in India we understand that to all intents and purposes patients are operated on in the open air, at least as compared to the closed rooms necessary in America and Europe. This free supply of air is important, whether we believe death to be imminent from cardiac or respiratory failure, but this supply of air matters little to the patient if he does not breathe freely, nor does the dose of chloroform amount to aught if it is not drawn into the chest. The dose of chloroform is not the amount on the inhaler, but the amount taken into the chest, and, finally, the amount absorbed by the blood-vessels. The rapidity and depth of respiratory movements is, therefore, as Lawrie asserts, the entire key to the situation.

We watch a windmill over a well to see if it is pumping into a reservoir a given quantity of water. If the windmill works irregularly, so that we know its pumping action is deranged, we separate it from the pump until it works steadily. Similarly we withdraw chloroform, as Lawrie says, whenever respiration becomes disturbed in rhythm, or when struggling disturbs it, because the first indicates that the drug's action is uncertain, and because there is no telling the dose which is absorbed. Whilst watching the respiration will not warn us of a sudden cardiac arrest in fatty heart plus chloroform depression, neither will the pulse give us such warning, and we are confident that the statement of the Hyderabad Commission, that the *respiration should be watched,* is correct, for we believe from a long series of observations, that gradual cardiac failure never occurs without producing respiratory changes from the very first. In other words, we do not believe that in a healthy heart chloroform can cause serious disorder without, as a result of beginning disorder, disturbance of the respiration; and, second, that in a healthy heart a quantity of chloroform sufficient to disorder it will, by its direct action, disorder the respiration. If, as an extra precaution, one assistant watches the pulse while the other watches the respiration, very well, for though the respiration is the more important function to watch, the man watching the pulse might discover an irregularity which the anæsthetizer may not see reproduced in the respiratory action, but as divided attention generally means a slighting of both objects in view, Lawrie is right in insisting on the pulse being let alone.

5. In answer to question 5 we have several points to offer.

(*a.*) Hot climates, where ether is inapplicable and where a free circulation of air increases the safety of the patient.

(*b.*) Chloroform may be used whenever a large number of persons are to be rapidly anæsthetized, so that the surgeon may pass on to others and save a majority of lives, even if the drug endangers a few, as on the battle-field, where only a small bulk of anæsthetics can be carried.

c. Its employment is indicated in cases of Bright's disease

requiring the surgeon's attention, owing to the fact that anæsthesia may be obtained with so little chloroform that the kidneys are not irritated, whereas ether, because of the large quantity necessarily used, would irritate these organs. Quantity for quantity, ether is, of course, the less irritant of the two.

d. In cases of aneurism, or great atheroma of the bloodvessels, where the shock of an operation without anæsthesia would be a greater danger than the use of an anæsthetic, chloroform is to be employed, since the greater struggles caused by ether and the stimulating effect which it has on the circulation and blood-pressure might cause vascular rupture.

e. In children or adults who already have bronchitis, or who are known to bear ether badly, or, in other words, have an idiosyncrasy to that drug, chloroform may be employed.

f. Persons who struggle violently, and who are robust and strong, are in greater danger from the use of chloroform than the sickly and weak, probably because the struggles strain the heart and tend to dilate its walls.

The safest method of administration is by Lawrie's or Esmarch's inhaler, because these provide free circulation of air and do not distract the attention of the anæsthetizer from the respiratory movement by complicated apparatus. Apparatus like these, in allowing a free amount of air, are the Hyderabad chloroform inhaler, or open-ended cone, with Krohne and Sesemann's respiration indicator attachment.

The Junker inhaler, even with its modifications, is too complicated and cumbersome, and while less chloroform is wasted in administering the drug, it must all be thrown out of the bottle afterward. If used at all it should be used with the increased air supply and respiration indicator of Krohne and Sesemann.

We agree so heartily with Lawrie's personal conclusions that we print them below.

"1. The chloroform should be given on absorbent cotton, stitched in an open cone or cap. (A depression made through the opening in the inside flannel bag will answer as well.)

"2. To insure regular breathing, the patient lying down

with everything loose about the neck, chest, and abdomen, should be made to blow into the cone, held at a little distance from the face. The right distance throughout the inhalation is the nearest which does not cause struggling, or choking, or holding of the breath. Provided no choking or holding of the breath occurs, the cap should gradually be brought nearer to, and eventually may be held close over, the mouth and nose as insensibility deepens.

"3. The administrator's sole object while producing anæsthesia is to keep the breathing regular. As long as the breathing is regular and the patient is not compelled to gasp in chloroform at an abnormal rate, there is absolutely no danger whatever in pushing the anæsthetic till full anæsthesia is produced.

"4. Irregularity of the breathing is generally caused by insufficient air, which makes the patient struggle or choke, or hold his breath. There is little or no tendency to either of these untoward events if sufficient air is given with the chloroform. If they do occur the cap must be removed, and the patient must be allowed to take a breath of fresh air before the administration is proceeded with.

"5. Full anæsthesia is estimated by insensitiveness of the cornea. It is also indicated by stertorous breathing, or by complete relaxation of the muscles. Directly the cornea becomes insensitive, or the breathing becomes stertorous, the inhalation should be stopped. The breathing may become stertorous while the cornea is still sensitive. The rule to stop the inhalation should, notwithstanding, be rigidly enforced, and it will be found that the cornea always becomes insensitive within a few seconds afterward.

"It is only necessary to add that the patient should be so dressed for an operation that his respiratory movements can be easily seen by the chloroformist. In the climate of India this is not difficult to manage, but it is more so in the climate of Europe; so that in this respect, and in this respect alone, the chloroformist in England is placed at a distinct disadvantage compared with the chloroformist in India."

NOTE.—Since writing this report two important papers upon this subject have appeared: the paper by Gaskell and Shore, in the London *Lancet*, in which they carried out a complete line of ingenious cross-circulation experiments, and from which they conclude that the fall in blood-pressure seen under chloroform is due to cardiac rather than vasomotor depression; and another paper, published by Lawrie in the *Lancet* for February 11, 1893, in which he refutes the statements made by Gaskell and Shore, and details experiments which combat those of the two investigators just named.

We cannot help believing that cross-circulation experiments in regard to the action of chloroform must be received with considerable doubt by the practising physician. Even if such work is carried out with the greatest skill the opportunities for error are innumerable, and while, if the results obtained were in accord with other studies, they might be accepted as confirmatory, the fact that they differ makes their negative conclusion of little value.

The object of the investigator of the action of chloroform is to perform experiments which, as far as possible, will be counterparts of the employment of the drug for human beings.

To the physiologist it is important to study a drug in order that certain results may be obtained, whether they have a practical bearing or not, but the practising physician only wishes to elucidate those points which should guide him in the administration of the remedy.

The concluding paragraph of Lawrie's latest contribution to the subject states the facts so clearly, and is in great part so in accord with what we have tried to set forth in our own report, that we cannot do better than quote it:

"The Hyderabad Commission's work proves that, while Syme's principles are right, there is no such thing as a safe method of chloroform administration. It is no longer a question of the superiority of the London method, or of the Edinburgh method; absolute safety can be attained neither by watching the respiration nor the pulse for signs of danger, which are in either case proof of improper administration or

of over-dosing. Moreover, over-dosing may take place whether the anæsthetic is given on lint or on a towel, or on a cap such as we use, or with Junker's, or Skinner's, or any other form of apparatus. The all-important point is that the breathing shall never be interfered with in any way. Safety under chloroform can unquestionably be insured, but only by regular natural breathing; and, whatever method is employed, no one can deny that it is the bounden duty of the chloroformist to maintain natural breathing throughout the whole administration. To maintain natural breathing requires careful training and considerable experience, but if this condition be fulfilled it is impossible to produce anything with chloroform but anæsthesia, and the Hyderabad Commission has shown that anæsthesia alone is entirely free from risk." [1]

In reply to a general request for reports of cases of accident under chloroform, we received the answers shown in the appended Table, which may be summarized as follows:

Respiratory failures	29
Reported unable to feel pulse while respiration continued	4
Simultaneous failure	1
Not stated	1
Total number of accidents reported	35

Of the 29 respiratory failures there were 5 deaths, a percentage of 17¼. Of the 4 circulatory failures there were 2 deaths, a percentage of 50. The one case of simultaneous arrest of respiration and circulation resulted in death.

This summary is particularly interesting in that the great majority of accidents were due to respiratory failure and not to the heart; and this failure was irrespective of age, sex, condition, or magnitude of operation. Also, that the accident may occur before, during, or after the operation; and finally, that in some instances circulatory failure takes place while respiration continues.

[1] Provided the drug is not pushed too far, and that the patient is in ordinary health. We would prefer to make the last sentence read "anæsthesia can be safely produced by chloroform." (Hare and Thornton.)

The *tracings* illustrating this research will be found in the Therapeutic Gazette for October, 1893.

Name of reporter.	Age.	Sex.	Operation.	State of patient at time of beginning anæsthetic.	Kind of chloroform administered	Anæsthetist.	How administered	Rapidity of administration.	Concentration of vapor.
.....	21 yrs.	M.	Oblique inguinal hernia.	Patient calm	Squibb's C. P. chloroform.	Skilled.	On towel.	Few drops at a time.	Plenty of air.
A. M. Hayden, Evansville, Ind.	67 yrs.	M.	Epithelioma of upper lip.	Patient calm	Chloroform.	Not stated.	Not stated	Not stated	Not stated
H. S. Harrington, Bertrand, Neb.	8 mos.	...	Talipes varus.	Not stated.	U. S. P. chloroform.	Skilled.	" "	" "	" "
T. F. Hallett, Rose, N. Y.	6 yrs.	M.	Amputation of cut finger.	Not stated.	Quality of chloroform not stated.	"	" "	" "	" "
W. H. Washburn, Milwaukee, Wis.	45 yrs.	M.	Renal colic.	Not stated.	Squibb's C. P chloroform.	"	On handkerchief.	Few drops at a time.	Plenty of air.
M. P. Murin, Denver, Col.	9 yrs.	M.	Tenotomy of tendo Achillis and plantar facia	Not stated.	Not stated.	Unskilled interne.	Not stated	Pushed as if it were ether.	Concentrated vapor of chloroform.
M. P. Murin, Denver, Col.	22 yrs.	M.	Tight stricture, internal urethrotomy.	Patient calm	Pure chloroform.	Not stated.	" "	Not stated	Not stated
M. P. Murin, Denver, Col.	45 yrs.	M.	Stricture.	Took drug badly.	Not stated.	" "	" "	" "	" "
E. Lamphear, Kansas City.	Not stated	Not stated	Cancer of pancreas.	Not stated.	" "	" "	" "	" "	" "
A. W. Wilmarth, Norristown, Pa.	16 yrs	F.	Epileptic convulsions.	Not stated.	" "	" "	" "	" "	" "
C. T. Southworth, Monroe, Mich.	Not stated	Not stated	Not stated.	Not stated.	" "	" "	" "	" "	" "
C. T. Southworth, Monroe, Mich.	Not stated	Not stated	Not stated.	Not stated.	" "	Skilled.	Esmarch inhaler.	Few drops at a time.	Plenty of air.
C. D. Westcott.	16 yrs.	M.	Sarcoma of	Not stated	" "	Not stated.	Not stated	Not stated	Not stated
.....	3 yrs.	F.	Hare-lip, cleft palate.	Not stated.	.....	.....	.....	.....	.....
J. H. Kellogg, Battle Cr'k, Mich.	24 yrs	M.	Left inguinal hernia.	Quiet.	Chloroform supposedly pure.	Not stated.	Junker inhaler.	Given carefully.	Not stated
L. Reynolds, Horton, Kas.	28 yrs.	M.	Enteralgia.	Quiet.	Squibb's C. P.	" "	Handkerchief.	Patient pulled drug over face and mouth.	" "
.....	5 yrs.	M.	Crushed finger.	Not stated.	Not stated	Unskilled.	Not stated	Given carefully.	" "

Chloroform to produce General Anæsthesia.

Length of time chloroform had been administered at time of accident.	Quantity administered up to this time.	First symptoms.	State of patient at time of accident.	Respiration and heart: which ceased first.	Time of accident.	Remarks.	Treatment.	Result.
Five minutes.	8 c.c. of drug taken.	Patient suddenly livid.	Struggling.	Respiration ceased several minutes before heart stopped	Operation not begun.	.....	Inverted and artificial respiration.	Death.
Not stated	Not stated	Sddenly respiration and pulse ceased.	Not stated	Both practically ceased at once.	Not stated	Has seen a number of non-fatal cases have respiratory failure, and resuscitated them	Inverted and artificial respiration.	Death.
" "	" "	Slight gasps given.	" "	Breathing stopped three minutes; heart continued to beat for this period after cessation of respiration.	" "	.....	.....	Death.
" "	" "	Patient stopped breathing.	" "	Breathing stopped; heart beating.	" "	Everything going well; drug withdrawn from two to four minutes before accident occurred.	Inverted and artificial respiration.	Recovery.
" "	" "	Respiration ceased.	" "	Respiration ceased; hut pulse continued.	No operation.	After first recovery chloroform again given, and accident repeated.	Artificial respiration.	Recovery.
Operation half over; time not stated.	Full quantity, exact amount not stated.	Respiration ceased.	Probably not struggling.	Respiration ceased; heart beating.	Operation halt over.	One-third grain sulphate of morphine was given before anæsthetic was begun.	Artificial respiration for 25 min.	Recovery.
Operation over; exact time it had beeu administered not stated.	Not stated	Face suddenly purple; eyes protruded.	Quiet.	Respiration ceased; pulse strong.	Operation over.	Resp. stopped as bladder was distended with irrigation.	Artificial respiration, inversion, nux vomica, belladonna, whiskey.	Recovery.
Operation not begun; time not stated.	" "	Spasm of muscles of respiration; respiration stopped.	Struggliug.	Respiration ceased; pulse good.	Operation not begun.	All muscles of respiration very spastic.	Inverted and artificial respiration.	Recovery.
Not stated	" "	Respiration stopped.	Not stated	Respiration stopped; pulse good for five minutes.	Operation begun.	Resp. stopped as finger touched diaph'm through incision in belly.	Artificial respiration and stimulation.	Death.
" "	" "	Not stated.	" "	Respiration stopped beforc heart.	Not stated	.....	.....	Death.
" "	" "	" "	" "	Respiration stopped before heart.	" "	Operator and others noticed that pulse continued.	.....	Death.
Operation nearly over; exact time not stated.	" "	Stopped breathing.	Quiet.	Respiration stopped; radial pulse good.	Operation over.	Operator and others noticed that pulse continued.	Not stated.	Death.
Not stated	Not stated, but large.	Respiration ceased.	Quiet.	Respiration stopped, hut pulse full and strong.	Operation not yet begun.	.....	Artificial respiration and inversion.	Recovery.
.....	.....	.....	.....	.....	.....	.....	Artificial respiration and inversion.	Recovery.
Late, operation almost completed	Not stated	Face asumed livid hue.	Not stated	Heart stopped; respiration continued for a few moments.	Operation completed	Particular attention paid to see which stopped first, heart or respiration.	.....	Death.
After a few moments.	" "	Respiration stopped.	" "	Respiration stopped; heart strong.	.....	.....	Artificial respiration.	Recovery.
Early in administration.	Not statcd	Pulse stopped respiration labored.	" "	Pulse stopped; respiration labored.	Operation not completed at time of accident	Same occurrence three times in same case before operation was completed.	Artificial respiration and stimulation.	Recovery.

Name of reporter.	Age.	Sex.	Operation.	State of patient at time of beginning anæsthetic.	Kind of chloroform administered	Anæsthetist	How administered	Rapidity of administration.	Concentration of vapor.
H. Mallens, S. O. Wattson, Norfolk.	2 yrs.	M.	Removal of cancerous testicle.	Not stated.	Not stated.	Not stated	Not stated	Given carefully.	Not stated
Robert T. Morris, New York.	4 yrs.	M.	Club-foot.	Took drug well up to time of full anæsthesia	"	"	"	Not stated	"
Robert T. Morris, New York	30 yrs.	F.	Dilatation of cervix	Not stated.	"	"	"	"	"
Robert T. Morris, New York.	35 yrs.	F.	Straightening a flexed uterus.	Not stated.	"	"	"	"	"
J. T. Webster, Emporia, Kas.	4 yrs.	M.	Paraphimosis.	Not stated	"	Unskilled.	On cloth.	"	"
J. T. Webster, Emporia, Kas.	Not stated	M.	Amputation of the thumb.	Not stated.	"	Not stated	Not stated	"	"
J. N. Coons, Palmora, Mo.	Young adult	M.	Removal of necrosed sequestrum of tibia.	Very weak and exhausted.	"	Skilled.	"	"	Well diluted with air
J. P. Hackenberg, Austin, Texas.	35 yrs.	F.	Operation on foot	Not stated.	"	"	"	"	Not stated
G. W. Shidler, York, Neb	38 yrs.	M.	Fistula in ano.	Not stated.	"	"	"	"	"
Louis J. Pons, Roxbury, Conn.	25 yrs	M.	Not stated.	Not stated.	"	Not stated	On napkin.	"	"
J. F. Baldwin, Columbus, O.	Eld'ly adult	F.	Removal of urethral caruncle.	Not stated.	"	Young physician	Not stated	"	"
A. W. Wilmarth, Norristown, Pa.	16 yrs.	Not stated	No operation—given to quiet epileptic convulsions.	Struggling.	"	Skilled.	"	"	"
F. Walter Todd, Redondo Beach, Cal.	Not stated	F.	Not stated.	Not stated.	"	"	"	"	"
Louis J. Pons, Roxbury, Conn.	6 yrs.	M.	Not stated.	Not stated.	"	Not stated	"	"	"
Thos. R. Savage, New York.	30 yrs.	F.	Operation upon rectum	As the patient was insane, it is probable she was not calm.	"	"	Esmarch inhaler.	"	Admixture of air.
Thos. R. Savage, New York.	24 yrs.	M.	Removal of catheter from bladder	Not stated.	"	"	Esmarch inhaler.	"	Admixture of air.
Thos. R. Savage, New York.	47 yrs.	F.	Carcinoma of breast.	Not stated.	"	"	Esmarch inhaler.	"	Admixture of air.
Thos R. Savage, New York.	7 days	M.	Spina bifida.	Not stated.	"	"	Esmarch inhaler.	"	Admixture of air.

Length of time chloroform had been administered at time of accident.	Quantity administered up to this time.	First symptoms.	State of patient at time of accident.	Respiration and heart: which ceased first.	Time of accident.	Remarks.	Treatment.	Result.
Five min.	Not stated	Respiration ceased.	Not stated	Respiration ceased.	Before operation.	The accident being repeated, no operation was done until next day, then under ether without difficulty.	Artificial respiration.	Recovery.
.....	" "	Stopped breathing.	" "	Respiration ceased.	Not stated	.....	Not stated.	Recovery.
Beforeoperat'n; time not stated.	" "	Cessation of respiration.	" "	Respiration ceased; pulse good.	Just before operation.	.....	" "	Recovery.
Not stated	" "	Stopped breathing.	" "	Respiration ceased.	Not stated	.....	" "	Recovery.
" "	" "	Stopped breathing.	" "	Respiration ceased; pulse continued good.	" "	....	Inverted and artificial respiration.	Recovery.
" "	" "	Stopped breathing.	Perfectly limp.	Respiration ceased; pulse full and regular.	During operation.	.....	Inverted.	Recovery.
" "	" "	"Pulse stopped for at least one minute."	Not stated	Pulse could not be felt.	Not stated	Thinks death would have resulted had he not closely watched the pulse.	Stimulated with ammonia, and fresh air admitted to room.	Recovery.
A few minutes.	" "	Stopped breathing.	Deathly pale.	Respiration.	After operation, and patient stood erect	Attributes the accident to allowing patient to regain her feet too soon after operation.	Artificial respiration and cold interrupted douche to chest.	Recovery.
Not stated	3 or 4 drachms.	Heart ceased to beat.	Not stated	Pulse could not be felt; resp. contin'd 1 min. longer.	Not stated	Patient had taken chloroform ten years ago for extraction of tooth.	Artificial respiration.	Death.
" "	Not stated	Not stated.	" "	Respiration.	" "	....	Artificial respiration, hypodermic injection of ammonia.	Recovery.
A few minutes.	" "	Respiration ceased, and heart feeble.	" "	Respiration; pulse good for some time.	" "	.....	Artificial respiration.	Death.
A few minutes.	" "	Respiration suddenly stopped; pulse good.	" "	Respiration	" "	.....	Artificial respiration.	Recovery.
A few minutes.	" "	Respiration ceased.	" "	Respiration.	" "	Same accident occurred twice in this operation.	Artificial respiration.	Recovery.
Not stated	" "	Not stated.	" "	Respiration.	" "	.....	Artificial respiration, hypodermic injection of ammonia.	Recovery.
" "	" "	Respiration ceased.	" "	Respiration.	Under full anæsthesia during operation.	The same result was experienced a month later for another operation upon her.	Artificial respiration and inversion.	Recovery.
.. ..	" "	Respiration ceased.	" "	Respiration.	Under full anæsthesia during operation.	.....	Inversion and artificial respiration.	Recovery.
Before anæsthesia was complete.	" "	Respiration ceased.	" "	Respiration.	Before complete anæsthesia.	The accident reoccurred several times, and as it was thought this was a case with idiosyncrasy to chloroform, ether was substituted with satisfactory result.	Artificial respiration.	Recovery.
Not stated	" "	Cyanosis and arrested respiratory movement.	" "	Respiration.	During operation.	.....	Inversion and slaps upon back.	Recovery.

DISCUSSION.

Dr. H. C. Wood: The paper of Dr. Hare is of interest from various points of view, and especially as showing how, under continued research, facts which seemed to be antagonistic drift into one another. If, however, we study the absolutely new light that is thrown upon the subject, we find that the evidence brought forward by Dr. Hare is of two characters. In the first place there is the negative evidence, and in the second place there is the positive evidence. The negative evidence is, that Dr. Hare has made so many experiments upon animals and has failed to see cardiac arrest, and upon this failure his extraordinarily positive assertion that death never occurs is based. The statement was also made by Dr. Hare that a great Indian surgeon had produced anæsthesia with chloroform in twenty-five thousand cases and had not had a death from cardiac arrest, and that, therefore, death does not occur in man from cardiac arrest; but the same gentleman had no death, so that reasoning from negative evidence to positive assertion, we have proof that death does not occur during chloroform anæsthesia. Negative evidence, in spite of positiveness of assertion, remains negative evidence.

The positive evidence which interested me very much is the proof that chloroform is a depressant to the heart, and that it is possible for this depression to go so far as to paralyze the heart under certain circumstances, as when it is placed directly in contact with the heart-muscle.

Another interesting piece of positive evidence is that which controverts what has been a strong point of the Hyderabad Commission, namely, that a needle put into the heart shows that the organ still moves although there is no pulse in the carotid. In my own experiments I have never had a needle in the heart. Dr. Hare has shown that the movement of the needle is no proof of effective cardiac beating.

Now, so far as the negative evidence is concerned, I simply state that I have made careful inhalations of chloroform—not injections—and I have seen the cardiac arrest precede the respiratory arrest by one minute or even longer; but I say, further, that this is an exceptional result and not to be seen very frequently. I have seen cardiac arrest nearly simultaneous with the respiratory arrest, but as the usual thing I have seen arrest of respiration first. The statement was made by Dr. Hare that the great fall of pressure is of little or no importance, and that the heart is not paralyzed because it restores itself under artificial respiration. I have seen the respiratory centre restore itself after its function had ceased for a minute and a half, so that arguing in the way Dr. Hare argues the conclusion is inevitable that chloroform paralyzes the respiratory centre. Both the respiration and the heart action will come back under the influence of artificial respiration.

All I claim is that it is an exceptional result for the heart to be paralyzed by chloroform before respiration ceases. An important practical evidence is

that we have had numbers of careful surgeons all over the world who have affirmed that they have had death in man from heart failure, and I do not see how that is to be gotten over. The experimental facts admitted by Dr. Hare, that the drug is a cardiac depressant and that it is possible to paralyze the heart, tally with the clinical observations, which show that it is possible under certain circumstances for the drug to act overwhelmingly upon the heart of the individual patient.

The matter, after all, is not of so much importance as it seems, for the simple reason that if you get death from respiratory paralysis under the influence of chloroform, you get it largely because there is so much cardiac depression. Although the last quiver of the heart does not occur before the final respiratory act, the heart is so depressed that the respiratory action of the drug becomes dangerous. Another important practical point is that there is an immense difference in the way that chloroform holds on to its victim as compared with ether. I have recovered animals apparently dead under ether by merely compressing the chest, but when once the respiratory or cardiac centre is stricken down by chloroform, it stays down, unless the resort to forced artificial respiration be very prompt and active.

Dr. Edward T. Reichert: So far as my experience has gone with animals under anæsthesia, I have certainly found that in nearly all fatal cases death has ensued from primary arrest of respiration. But the effect on the heart is different under chloroform and under ether. Where respiration and heart-beat have ceased in ether anæsthesia, the heart's action may return, but in chloroform anæsthesia when the heart stops, it stops forever. In other words, there is a probability of the restoration of the heart's action when it has been arrested by ether, but practically none in chloroform poisoning.

Dr. Frank Woodbury: The late Professor S. D. Gross told me that he had administered chloroform upward of ten thousand times without a single death. His special instruction to the anæsthetiser was to watch the pulse; but I have frequently seen him stop an operation and call attention to the fact that the patient was breathing badly, and immediately institute measures to resuscitate him, thus showing that he also watched the respiration. The results of the large experience of Professor Gross are, therefore, entirely in support of the position of Dr. Hare, that both the respiration and the pulse should be watched, more especially for the reason that we can never tell whether or not the heart is in an exactly normal condition.

As to the relative safety of ether and chloroform, I wish to make a single observation: it is that the ethyl and methyl compounds exert different effects upon the higher nerve-centres. This difference is radical, and is analogous to that between sodium and potassium salts upon the heart. The compounds of ethyl act as cardiac stimulants; the oxide of ethyl can, therefore, be administered with perfect safety during a prolonged period. On the contrary, chloride of methyl is a depressant and can only be administered for a short time; it is especially serviceable for the class of operations referred to in the

paper where rapid anæsthesia is desired, or it is necessary to operate on a large number of persons in a short period of time.

The author of the paper referred only to the medullary, vasomotor, and cardiac centres. I should like to call attention to the fact that the higher or cortical centres may also deserve some consideration. Man is a highly emotional creature; he is also highly developed intellectually, and in his daily life all his actions are ordinarily dominated by his emotional and intellectual nature. We know that the direct effect of fright may be to depress the circulation, blanch the face, weaken the pulse, cause syncope and even death. Why does this not always occur? Simply because, in most instances, by the exercise of the power of the will the cortical centres exert an inhibitory influence upon the lower centres in the medulla. My explanation of the suddenly fatal action of chloroform, at least in some cases, is that it temporarily abrogates consciousness and will-power by its depressing effects upon the upper centres, and so removes this inhibition; and, therefore, should syncope occur the patient is liable to perish owing to the attendant depression of the medullary centres to which Dr. Hare has referred, and which prevents the prompt restoration of the heart's action by the usual means for overcoming syncope. The fact that inversion of the patient is a recognized means of successful treatment of chloroform narcosis is in support of this view; as is also the engorged condition of the cavities of the heart found at the autopsy of such cases of sudden death during the primary stage of chloroform anæsthesia. Both of these explanations, it seems to me, deserve consideration in any discussion of the value of chloroform, and of its comparative safety with other agents employed to produce temporary anæsthesia. The latter explanation may also throw some light upon the question of why chloroform may exert different effects in man from those observed in the dog.

Dr. Hare: Dr. Wood misunderstood me in regard to the action of chloroform. I did not say that chloroform death was never due to cardiac failure. Lawrie practically has said that. I said that I had not known it to be so, and I am not willing to go on record as stating dogmatically that it never occurs.

In regard to the statement that great surgeons have given chloroform many times and asserted that death was sometimes due to cardiac failure, the fact is that the great surgeons have not had the deaths, but the residents in the hospital, and, as in two cases that I have seen, the surgeon has not known that the patient was doing badly until he was dead, and then the attempt to ascertain whether the heart or breathing stopped first was impossible.

There is another point: you will frequently find the peripheral arteries empty, but on opening the chest you will find the heart beating and supplying blood to the large organs and to the brain. There seems to be a condition in which the circulatory system recognizes that it is impossible to supply blood to every part, and the blood ceases to go through certain unimportant vessels.

When we find the pulse in this condition and open the chest rapidly, the heart is found widely dilated and abortively beating. If the hand is put in the chest and the heart squeezed so as to help it a little, in two or three minutes the heart is going on as well as ever. It is not a true cardiac depression, it is first a cardiac dilatation. If the heart is capable of ridding itself of this blood, it can go on. If it cannot, the efforts become more feeble and the heart stops from paralytic distention.

Dr. Wood: If this explanation be correct, that the heart is arrested in this condition of dilatation in the human death from chloroform, what is the reason that so many clinicians have found that if the patient be suspended with the head down the heart will start up? Placing a man on his head fills the heart full of blood from the great abdominal vessels; and it does not seem possible for the blood to run out of the right heart, at least, on account of the congested and obstructed state of the lung circulation.

Dr. Hare: I do not know that I can answer Dr. Wood's question. In most of these cases the death is respiratory, and by turning the patient upside down you send fresh blood to the respiratory centre. I have found, contrary to what Dr. Wood has stated, that if you invert an animal the blood will run out of the great vessels at the base of the heart instead of into them, and if the reverse were true, the valuable service rendered by inversion would not exist, as the heart, already depressed, would be more than ever overloaded.

CASE OF AN ENORMOUS ANEURISM.

By J. K. MITCHELL, M.D.

[Presented June 7, 1893.]

The interesting specimen, which I am glad to have the opportunity of showing to the College in its fresh state, was removed yesterday from a patient who was first seen on the 5th of last February by Dr. S. Weir Mitchell and myself. He was fifty-five years old, and, as may be seen, had an enormous aneurism springing from the upper part of the transverse portion of the aorta. It began in the middle line and grew directly upward, or rather from the upper wall, pushing aside and backward the left carotid and innominate arteries, so that the former lies over its left edge considerably back of the usual position, while the latter is entirely behind the mass of the tumor. The trachea, too, is pushed around to the left and back of the aneurism. The latter presses upon the trachea, the œsophagus, and the great vessels, but does not seem to cause any decided pressure upon the nerves. The relation of the parts is not only curious, but, when compared with the clinical signs during life, exceedingly interesting.

The patient presented absolutely no direct signs of aneurism except a difference in the two radial pulses. There was an area of dulness in the upper part of the chest, which was considerably larger when he was first seen in February than it was just before death. There was almost complete obliteration of the supra-sternal notch. The vessels on both sides, above and below the clavicle, were greatly enlarged—some of them as big as my little finger—and very much congested. The patient had a cough, but not of the character usually associated with aneurism. There was considerable difficulty in breathing on slight exertion, and a wheezy sound could be heard all over the chest, obviously caused by pressure upon the trachea. His pulse was not noticeably affected, save that the left one was smaller and feebler than the right; although this suggested aneurism, there was no bruit, nor did there seem to be any dilatation of the aorta.

The patient was put to bed, kept upon a full milk diet, and large doses of iodide of potassium were given on the supposition that the growth in the

chest was a solid one and of specific origin. From the first week he steadily improved. The area of dulness in the upper portion of the chest diminished to about one-third of its original extent, the swelling of the neck lessened by nearly two inches, the vessels returned to their normal size, the dyspnœa disappeared, the whistling breathing lessened very greatly in degree, and for a month before his death was not to be heard, except immediately over the presumed situation of the trachea and upper bronchi.

An imprudent exposure of himself when he was already out of bed and gradually getting about, brought on œdema of the lungs, which, in spite of active treatment, grew speedily worse, and he died on the fourth or fifth day.

We found, at the post-mortem, that the aneurism was entirely healed, or, if you like, let us say cured. It was perfectly solid, and presented no other appearance than that of a solidified tumor. Besides the extensive œdema, there were numerous small islands of semi-consolidation in the lungs, which were rather unusual in their wide distribution.

DISCUSSION.

Dr. H. A. Hare: I have had two or three cases in hospital and private practice which have closely resembled the one reported to-night. One case was that of a man, aged sixty-two years, in St. Agnes's Hospital, whose only symptom was curious laryngeal attacks, which came on during the day and which resembled in character paroxysms of asthma, but there were none of the physical signs of asthma. The most careful examination failed to reveal any sign of aneurism. There was no thrill or bruit. One day Dr. Dercum happened to be in the hospital and I asked him to see the patient, but the man gave his last gasp just as we reached his bed. At the post-mortem we found a curious condition. There was an enormous aneurism which grew entirely backward and which had a very large laminated clot in it. In the skull we found, over the motor area, an exostosis two and a half inches in length, one inch in width, and fully one-half inch in depth, which had displaced the convolutions, but had produced no cerebral symptoms.

Another case is of interest as illustrating that we may give the iodides or other remedies the credit of the cure, when in reality other causes have been at work. The patient was from New Mexico, and presented dyspnœa and all the symptoms of intra-thoracic growth without any of the ordinary signs of aneurism. After careful study it was concluded that it was a case of aneurism, and the history that the symptoms had followed a strain from lifting a wagon, favored this view. The man was put on iodide of potassium and improved wonderfully. Finally it dawned on us that the man improved not because of the iodides, but because he was at a lower altitude. He continued

to improve and left the hospital and went to Atlantic City, and still is in fairly good condition.

These cases emphasize the fact that bruit, thrill, and pulsation cannot always be detected in aneurism, and that aneurism with or without these positive signs is, after all, the most common form of intra-thoracic growth.

Dr. James Tyson: It occurs to me that the temporary aphasia, pointing directly to cerebral embolism, might have called attention to the fact of circulatory trouble.

CLEFT PALATE, BOTH HARD AND SOFT, AND HARE-LIP.

By W. S. FORBES, M.D.,
PROFESSOR OF ANATOMY, JEFFERSON MEDICAL COLLEGE, AND CLINICAL SURGEON TO JEFFERSON MEDICAL COLLEGE HOSPITAL.

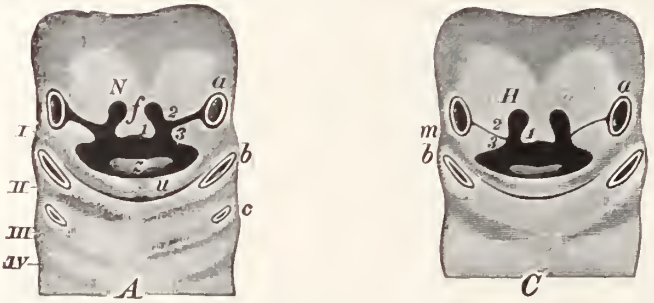
[Read April 5, 1893.]

The affections named in the title are congenital. At the end of the third week in the development of a human fœtus three deep pits, lined by a stratum of columnar epiblastic cells, appear on each side of the head, and ultimately coming in contact with outgrowths from the developing brain give rise to organs of special sense. One of these, the posterior, becomes the auditory vesicle; another, the middle, becomes the optic vesicle; and the third, the anterior one, placed near the median line, becomes the nasal vesicle. It is to this last that I wish to call attention.

The nasal vesicle, placed in front of the auditory and optic vesicles and close to the middle line, is connected by furrows with a deep invagination of epiblast, pushed in on the middle line, known as the stomodeum. At the fourth week of fœtal life, the border of the heads that forms the upper edge of the oral invagination below the anterior brain-vesicle, presents five marginal facial processes (Fig. 1, *A*). The central one of these processes is the single fronto-nasal process (*f*, Fig. 1, *A*), which is separated on each side from the lateral nasal process (2, Fig. 1, *A*) by a nasal invagination. External to the lateral nasal process is the ocular invagination at the outer and inferior border of which an outgrowth from the mandibular fold, the maxillary process, grows forward, separating the ocular invagi-

nation from the stomodeum. As it extends, its inner edge touches and coalesces with the outer side of the fronto-nasal process, making thereby a continuous upper lip for the aperture of the mouth. If from any cause the coalescence of the

Fig. 1.



Scheme of formation of the face, and arrest of the development. (From Landois.)

A. First appearance of the face; *I, II, III, IV*, the four visceral arches; *f*, fronto-nasal process; *N*, nasal orifice; 1, inner, and 2, outer nasal processes; 3, superior maxillary process; *u*, inferior maxillary process; *b, c*, first and second visceral clefts; *a*, eye, *z*, tongue.

C. Arrest of the development constituting oro-nasal cleft. H, Nasal orifice; 1, inner, 2, outer nasal processes; 3, superior maxillary process; *a*, eye.

maxillary process with the fronto-nasal process is arrested and does not take place, the deformity known as hare-lip is the result.

The union of the fronto-nasal and lateral nasal processes internally completes the edge of the anterior opening of the nostrils, while the thickening of these processes forms the nose. The lower edge of the oral invagination is made by the mandibular fold, which lies in front of the first visceral cleft, and in which the lower jaw, or mandible, is subsequently formed. These partitions between the nasal invagination and the mouth are at first superficial, but an ingrowing process from the maxillary process on each side eventually extends inward like a shelf to meet its fellow of the opposite side medially, thereby completing the roof of the mouth. At the same time the vertical septum, which grows down from above, separates the nasal fossæ and forms the nasal septum. The posterior extension of the middle nasal process, which takes part in the formation of the palate, becomes the premaxillary process.

Failure of union of the processes at the middle line taking place, or their development being arrested, the deformity known as cleft palate follows.

The failure of this median union of these processes may be partial or complete. Thus, the want of median union of the

FIG. 2.

maxillary processes posteriorly only will produce a cleft of the soft palate; or a failure to unite may extend further, thus producing a cleft in the hard as well as in the soft palate; or the failure in development may be complete and no union exist between the lateral nasal process of one side and the proximal surface of the fronto-nasal process, thereby producing a continuous cleft through the lip and through the hard and soft palate. When the failure to develop is on both sides of the fronto-nasal process, then we have a double hare-lip and a V-shaped fissure through the vault of the mouth, and between the bifur-

cations of the V we find the undeveloped premaxillary nuclei, in which the incisor teeth are in time developed.

The failure in development and in attachment on both sides of the fronto-nasal process produces a hideous deformity. The patient I now bring before you presents such a condition.

C. A., a farmer's boy, nineteen years of age, coming from Virginia, presented himself to me in January, 1892; he was in reasonably good health;

FIG. 3.

he had a double hare-lip, and a wide cleft in the soft and hard palate. The cleft bifurcated anteriorly, each bifurcation following the maxillo-premaxillary sutures. The vomer was greatly developed. The lower anterior marginal thickening coalesced with the premaxillary bones, which were pushed forward and upward. These parts were covered by a dense mass of vascular connective tissue. The skin did not cover the anterior surface of the nasal septum, but projected forward, appearing like a well-developed nipple. There was an opening into each maxillary antrum. The entire lateral bony walls of the oro-nasal cavity were covered with a thick and very vascular integument, mucous membrane, connective tissue, and periosteum. The excessive vascularity in the whole field of operation was made very manifest in each operative procedure.

After several careful examinations, and after casts, diagrams, and photographs had been taken, I determined to first freshen the margin of the soft palate and then take lateral laps containing periosteum from the lateral walls and suture these flaps together in the middle line as far forward as the

maxillo-premaxillary sutures, leaving the treatment of the double hare-lip and the premaxillary bones and the greatly developed vomer for a future occasion. The antero-posterior diameter of the oral cavity was observed to be greater than usual. On the right side, the canine tooth, the two bicuspids, and two molars were well developed, but they were placed in a straight line from before backward and outward. On the left side the second molar was absent, but the teeth on the left, like those on the right side, ranged in a straight line backward and outward, the two lines forming a V-shaped figure

FIG. 4.

from before backward. The premaxillary bones with the incisor teeth were developed much in front of the apex of the V-shaped dental figure. Each half of the bifid uvula was larger than a normal uvula. The tensor palati and the levator palati muscles appeared to act with considerable force. The anterior pillars (palato-glossi) and the posterior pillars (palato-pharyngei) appeared to be subnormal.

A solution of cocaine was injected into the soft palate and into the parts covering the hard palate of each side of the fissure. The half-uvula of one side was seized with a long rat-tooth forceps and transfixed with a small double-edged, spear-shaped bistoury, and the incision carried up to a little nodule of bone, representing the undeveloped horizontal process of the palatine bone. A similar incision was then made upon the opposite side. Then, with a rectangular hoe-shaped knife, an incision was made along the lateral

wall, and on a lateral ridge representing the undeveloped palatine process of the maxillary bone, and carried as far forward as the anterior edge of the nasal process of the maxillary bone and its juncture with the ala of the nose. The soft parts, including the periosteum, were brought down nearly to the margin of the alveolar process, thus leaving the flap attached by a large and broad pedicle. The same procedure was carried out on the opposite side. The hemorrhage was very considerable, but was arrested by ice and pressure. It required nine silver-wire sutures to hold the margins of these flaps in

FIG. 5.

proper apposition in the middle line. The placing of the sutures was accomplished by means of a long needle with an eye at one end. This needle was thrust through the flap about one-quarter of an inch from its free margin and threaded from within, the wire being brought through the flap on the withdrawal of the needle, the needle then being thrust through the flap of the opposite side and threaded with the other end of the wire; then, withdrawing the needle, the wire was left in position. On drawing the edges of the flaps together a shot having a hole in it was run down the wire suture and compressed; the flaps were thus held well together. The making of the floor of the nose and the roof of the mouth was well accomplished by this procedure.

On washing out the nasal fossæ, none of the solution passed through the nasal floor to the mouth. The tension of the soft palate was now relieved by an incision on each side from above downward and obliquely, and at the

same time from before backward diagonally through the soft palate. Pledgets of sterilized gauze were then placed in the nose and cotton over the middle facial zone. This completed the operation for the time, and the patient was put to bed, and rectal alimentation was directed, with an anodyne.

After twenty-four hours had elapsed severe hemorrhage occurred, following a fit of coughing. After considerable difficulty this was arrested, and although the prostration was pronounced, the patient reacted rapidly

On the fourth day three of the anterior sutures were removed and union was found to be complete. On the sixth day the three posterior sutures were removed and here union was also found to be complete. On the seventh day the remaining three sutures were taken out. It was found that one of them had cut out. It was from this spot that the bleeding had taken place. There was found to be an opening the size of a pea, where the suture had cut through. With the exception of this little opening, the union was perfect throughout and the man was much improved. He went home in March. On his return, the last of January of this year, nearly ten months after the first operation, the parts were found to be well consolidated, and the small opening in the roof of his mouth had contracted a little.

While the fissure in the hard and soft palate was now closed, the facial deformity was still as great as ever. The second operation was performed early in February. Cocaine was injected into each lateral half of the lip. The parts were then dissected up from their attachment on each side to the maxillary bones, including the alæ of the nose. It was found, however, that these flaps could not be made to cover the premaxillary bones; nor could these bones be forced back into the narrow cleft between the maxillary bones and into the narrow apex of the V-shaped figure already spoken of. With my bone-forceps the premaxillary bones and that portion of the vomer in front of the gap were removed. The anterior margin of the septum that I had made some months before was now freshened, and the canine teeth having been extracted, a rectangular flap from each side, having a broad pedicle carrying the periosteum, was raised and made to meet in the middle line in front of the lower angle of the vomer. These flaps were held to the vomer and in apposition to each other by animal sutures. The lip flaps were now brought together in the middle line, and the parts were held in apposition by three steel pins.

The first pin was inserted into the cheek about a quarter of an inch external to the left ala nasi and passed nearly through the flap. It was then carried directly through the bony nasal septum and made to enter the right flap and reappear on the right cheek about a quarter of an inch external to the right ala nasi. This pin held the flaps well together and in excellent apposition. A second and a third pin were properly passed through the lip flaps. A well-sterilized silk ligature was now thrown around each individual

pin in the manner of a figure-of-8, and a sterilized gauze dressing was placed over the naso-facial zone.

The pins were withdrawn on the fifth day, leaving the ligatures *in situ* for the time being. On the seventh day when all the dressing was removed the

FIG. 6.

parts were found entirely united. A fortnight after the operation a dental plate was adjusted, containing the incisor teeth. A month after the last operation the man gave us the photograph which is here reproduced.

SYNCHRONOUS AMPUTATION OF BOTH THIGHS FOR RAILROAD INJURY.

By ISAAC MASSEY, M.D.,
WEST CHESTER, PA.

[Read September 6, 1893.]

Benjamin B., fifty-one years of age, colored, farm laborer, on the evening of March 4, 1893, was run over by an engine, crushing both legs from the knees down. It was at least one hour from the time that he was hurt until he was found. He was then carried into the baggage-room at the railroad station, where I was hurriedly called to see him.

His pulse was very feeble, irregular, and only about forty to the minute, his surface very cold. He was taken at once to the Chester County Hospital in West Chester, and at that time showed no evidence whatever of reaction. He was given $\frac{1}{8}$ grain of morphine with $\frac{1}{150}$ gr. of atropine hypodermatically.

An amputation of both thighs about eight inches above the knees was performed as speedily as possible, without waiting for reaction, with the assistance of Drs. Dunn and Patrick, and Joseph Scattergood, a student of medicine at the University of Pennsylvania.

Brandy and strychnine were frequently given hypodermatically during the operation, the latter in $\frac{1}{40}$ grain doses. When placed in bed his temperature was 96°, pulse 50, small and feeble. Under the use of artificial heat, in the form of hot water bags placed around him, and hypodermatics of $\frac{1}{40}$ grain of strychnine every four hours, with stimulants administered freely, he slowly reacted from the shock. His temperature at midnight, two hours after the operation, was 96°. At two o'clock it was 96.2°, at four o'clock it was 96.5°, and at five o'clock it was 98.5°. The highest temperature was on the third day after the operation, when it rose to 100.5°.

The dressings were removed on the seventh day. The right stump had united throughout without suppuration. There was slight sloughing of the posterior flap of the left stump, owing to the soft parts being severely contused, but he made a rapid and satisfactory recovery.

It is my opinion that the satisfactory results in this case should be attributed to the promptness with which he was taken to the hospital and the operation performed. It certainly supports the advisability in severe injuries of operating at once without waiting for reaction. The pulse slowly improved from the time the anæsthetic was commenced, and the operation in no way increased the shock already present, as it was formerly supposed to do.

A CASE OF WELL-MODULATED VOICE WITHOUT ARTIFICIAL APPLIANCE, IN A PATIENT FROM WHOM THE LARYNX AND FIRST TRACHEAL RING HAVE BEEN REMOVED, AND IN WHOM RESPIRATION IS MAINTAINED WITHOUT A CANULA.

By J. SOLIS-COHEN, M.D.

[Read October 4, 1893.]

The interesting case herewith presented is a remarkable exemplification of the admirable manner in which Nature, even when let alone, is sometimes fully competent to restore an important function after deprivation of the special structures normally employed in the exercise of that function.

A few days over eighteen months ago, on April 1, 1892, at my clinic in Jefferson Medical College Hospital, this man's larynx and the first ring of his trachea were removed for adeno-carcinoma protruding externally; but the epiglottis was left in position, as it was uninvaded by the growth. On the 25th of May following, the case was reported to the Philadelphia County Medical Society and the patient was exhibited wearing a canula; the orifice of his trachea presenting externally, as you see it now, and completely shut off from communication with the pharynx and the mouth.

Since then the man has learned to speak in a hoarse but fairly well modulated voice, audible, under favorable conditions, at more than forty feet. He is able even to turn a tune; and this without any artificial appliance. He does not even wear a canula.

For several months after the operation he was unable to speak, and was compelled to communicate his thoughts in writing; but one day when he was very much agitated he tried to talk to me, and made quite an audible noise in his throat. I encouraged him to make systematic efforts at reproducing the noise, so as to utilize the sound for speech; this he has done with such success that one might almost fancy a larynx to be an appurtenance as unnecessary as a vermiform appendix.

You will notice that when he wishes to speak he distends the skin of the neck above the trachea by compressing air into it as he swallows the saliva, so that it resembles in miniature a balloon, or, as Dr. Forbes has suggested, the bag of the Scotchman's bagpipe, then he lets the air strike in a staccato manner against some structures in his throat to produce the sound. I have thus far been unable to identify the structures even in the reflected image. The reflection in the mirror discloses a smooth funnel-like cavity, at the bottom of which is seen the transverse orifice of the œsophagus. The sound appears to come from just below this point, and the escaping air sets in commotion a layer of saliva on the left side, communicating a peculiar character to the voice. The patient insists that this saliva is essential to his best efforts. It seems to be a lubricator facilitating the movement of the new vocal reeds. As the sound can be modulated, the factors may be presumed to be the horizontal fibres of the inferior constrictor muscles of the pharynx and not some fortuitous folds of tissue which, if favorably located, might well produce a monotonous sound, such as that from the reed in a toy but incapable of modulation—for modulation requires the tension and relaxation of some elastic or contractile tissue.

For the normal production of voice, vocal bands are necessary; yet here is a man who has no larynx but who is able to produce a modulated voice audible at forty feet, by utilizing the constrictor muscles of his pharynx. The propulsion of air from the lungs is a physiological factor in normal phonation, but this man's lungs are entirely shut off from his pharynx, and he improvises a lung, so to speak, by distending the skin of his neck

with air into a sac, from which it is propelled in puffs which set his approximated pharyngeal constrictor muscles in vibration. Thus the mechanical requirements for voice are reproduced in contractile vocal reeds capable of approximation, tension, and relaxation; and in a contractile reservoir of air which can be discharged at will to set their edges in vibration; and this voice is broken into speech by the ordinary factors of articulation, the tongue, the lips, and the teeth. I have never seen a patient wearing an artificial larynx, but am told that this man's voice far exceeds in strength and in modulation anything that has yet been accomplished by the use of such a contrivance. It is certainly better than the voice of many patients with tumor, or tuberculosis, or stricture of the larynx; or even with chronic syphilitic laryngitis.

Let me distinctly disclaim any credit in purposely effecting the restoration of voice in this instance. That result has been the unexpected outcome of two preeautionary measures; one, a measure of security taken in the operation, and the other a measure of prophylaxis practised in the after treatment.

As is well known, a very large number of patients perish promptly from the effects of laryngectomy in consequence of septic pneumonitis excited by aspiration of secretions and excretions from the mouth and pharynx; and this, too, in some instances, despite the greatest care taken with the dressings.

While rehearsing the operation upon the cadaver, the day previous to its performance upon the patient, I discussed with Prof. Forbes the various measures which would best prevent this dreaded septic pneumonitis. Dr. Forbes suggested that I split two or three of the rings of the trachea anteriorly, and stitch them to the skin-flaps, so as to draw the top of the divided trachea forward and thus shut it off as far as practicable from the main wound. This was done. It was not neeessary to incise the trachea, for when the first ring was cut away from the second one, the lips of a tracheotomy incision which Dr. Forbes had made for me some weeks previously, gaped asunder; and these lips were carefully stitched to the skin, so as to tilt the orifice forward. You see the result. The divided end of the

trachea presents forward instead of upward, and its entire lumen opens out into the atmosphere so freely that in its present condition a canula would be but an actual encumbrance unless needed to diminish the supply of air. The patient carries a canula in his pocket, to be the better equipped should any emergency arise which might necessitate its reintroduction.

The second point is that no attempt was made to introduce an artificial appliance to take the place of the larynx; and chiefly so, because of the wish to keep the wound free from all unnecessary irritation until the danger of immediate recurrence should have passed. Before that period was reached, however, Nature announced vocally that such an appliance would be superfluous. Let alone to take care of themselves, for no dressing whatever had been applied to the interior of the wound, the structures have become so readjusted that the vocal function has been gradually reproduced by new factors. So far there has been no indication of recurrence of malignant disease. The patient is as happy as a poor man in need of light work can be; that is to say, he has no pain, no cough, no dyspnœa, no dysphagia, and no aphonia. Not long ago, he met at my office a man upon whom I had performed a prophylactic tracheotomy, but in whose case laryngectomy did not appear justifiable. That man has been relieved only of his dyspnœa. He retains his dysphagia, his cough, his pain, and his aphonia. The man without the larynx expressed the opinion that he was by far the better off; and stated that he would not care to live at all if he had to carry again his tumor, his larynx, and his canula.

That stitching the trachea to the skin increases the prospects of recovery of the patient after laryngectomy has been well shown in the special experience of Bardeleben (*Arch f. Chirurgie* Bd. 41; cited by Poppert, *infra*).

Bardeleben ("Vorträge zur Kehlkopfextirpation") closes the communication between the trachea and pharynx, and keeps it closed until the wound has become covered with good granulations, and until complications are no longer imminent; say for fourteen days. Then he removes the stitches in order to make

a communication for the insertion of an artificial larynx. Since adopting this method he has had four recoveries in succession, while under the ordinary management he had lost four patients out of five from lung complications.

Prof. Poppert, of Giessen, recently reported (*Deutsche med. Woch.*, 1893, No. 35) a case in which, in August, 1892, he performed an operation similar to that practised in the patient before us, but differing somewhat in detail. The trachea, however, was stitched to the skin and cut off entirely from communication with the pharynx. Immediately after the operation the patient was able to make himself undersood in whispers similar to those made by patients with occluded larynges. He was able to swallow liquid nourishment at once. The voice improved in strength, but still retained its whispering character at the time of the report, one year later.

Dr. Hans Schmidt reported, in 1888 (*Arch. f. klin. Chir.*, No. 38), a case of laryngectomy in which the trachea had become eventually cut off from communication with the pharynx after the patient had abandoned his medical attendants, who had vainly endeavored to keep the communication permanently open, and in which by the patient's unaided efforts, a rough, monotonous, unmodulated voice, had been ultimately produced, somewhat like the hoarse laryngeal whisper of laryngitis, but amply sufficient for practical purposes. (Landois and Stirling—*idem*.)

In the light of the results in the present instance and of the reports of the few somewhat similar instances on record, it may be advanced that stitching the trachea to the skin after laryngectomy, so as to shut the air passages off from communication with the pharynx, will increase the patient's chances of recovery from the operation by guarding him against most of the risks of direct septic pneumonitis. Furthermore, the free opening of the trachea forward will render a tracheotomy canula unnecessary, except, perhaps, for the first few days, in which it may be found desirable to occlude the trachea with a tampon surrounding the canula or jammed upon it. Finally, the adjustment of the parts may be left to Nature, with some prospect

of an eventual reproduction of voice, without the intermediation of any artificial appliance whatever; thus keeping the wounded structures free from irritation by a foreign body, and lessening the prospects of recurrence. Certainly, should there be occasion for another laryngectomy in my practice, the steps prudentially taken in the present instance would be purposely repeated, in the expectation that an intelligent furtherance of the natural processes of repair would favor and even promote an equally gratifying sequence.

Let us hope that the artificial larynx has had its day; and that it may no longer be regarded as an indispensable requisite for phonation after laryngectomy.

DISCUSSION.

Dr. W. S. Forbes: The factors of the organ of speech in this man have been separated by the division of the trachea and the removal of the larynx; those below the line of the divided trachea are lost to him so far as the organ of speech is concerned, the air entering and going out of the open end of the trachea above the sternal notch solely for the purposes of respiration.

The framework and body of the larynx, the organ of voice, having been removed by severance of its extrinsic ligaments, the broad and elastic thyro-hyoid membrane, the two lateral thyro-hyoid ligaments and the hyo-epiglottic ligament, the union of the margins of these and other soft parts has hermetically sealed the passages here from air from the pharynx.

The pharynx remains; its framework, its muscular walls except the origin of the inferior constrictor, which has been divided, its nerves and its blood-vessels are not injured. Its extensive attachments above to the base of the skull, below to the œsophagus, to the styloid processes through muscles on either side, to the internal pterygoid plate and pterygo-maxillary ligaments, to the lower jaw and to the tongue anteriorly are just as complete as before the operation. Its function as a portion of the alimentary canal is entirely uninjured. As a factor in the function of speech in this man, its agency is very limited, but important—without it he could make no sound.

The air enters his pharynx only by deglutition, and the amount is limited. It can escape into the œsophagus only to a very limited degree. The pharyngo-œsophageal orifice is surrounded by muscular fibre under the control of his will and capable of being brought under better management by practice.

His pharynx now may be compared to the bag of a Scotch bagpipe. He can pitch his sound only on one note, whether he sings or speaks, and can continue it at present just so far as to count nine, and his words can be distinctly heard at the distance of forty feet.

I believe this man will further improve in his power of speech. By practice he will gain further control over the muscles that play upon the hyoid bone, which, on account of its mobility and its elasticity plays such an important part in the organ of voice of everyone.

He will by practice obtain better control over the stylo-hyoidei muscles, and especially the individual fasciculi of the middle constrictor muscle, which has its origin entirely from the hyoid bone, thus allowing him to contract or relax parts of this muscle at will.

Of course the improvement can only be to a limited extent, but this is much to him.

The operation of removing the entire larynx on account of a morbid growth could not have had a happier issue.

Dr. Charles W. Dulles: I would ask if any measurements have been made as to the capacity of the cavity from which this man speaks, and also how many words he can speak without replenishing the air?

Dr. Cohen: No; but he can count up to "eight" or "nine" continuously without refilling his pharynx; but he appears when excited to refill the air chamber with each articulation. The limit seemed to be caused by the fact that the thorax remained immovable while the man continued an interrupted series of articulations, and that he had to pause in speaking when he inflated his lungs for respiratory purposes.

Dr. Dulles moved that the case be referred to a commission for investigation, mainly from a physiological standpoint.

The Vice-President, after consultation with Dr. Cohen, appointed Dr. Harrison Allen to investigate and report on the case.

CHLOROFORM ANÆSTHESIA.

By H. C. WOOD, M.D.

[Verbal communication, November 1, 1893.]

Mr. Chairman and Gentlemen, Members of this Society: It is with a good deal of reluctance that I have come forward to-night to say what I am about to say, becaue I had thought long ago that I was done with this controversy concerning anæsthetics. I had said what I believed to be the truth, and was willing to leave the matter to time and to the research of others to sift out the false from the true, knowing that what I had said that was good would remain, and that that which was evil had better perish. Circumstances with which you are familiar seem to demand that I should say something more, although covering somewhat the old ground. Especially have I been led to do this on account of the great confidence and respect that I have in so-called physiological therapeutics, for I believe that the only pathway along which medicine can travel to certain knowledge is the path of animal experimentation. It seems to me, therefore, that it is exceedingly important that certain principles of interpretation of animal experiments should never be lost sight of.

I propose to take the paper of Dr. Hare as a sort of a text on which to hang my thoughts. I shall first read certain conclusions which, although not the conclusions of Dr. Hare, but those of Surgeon Lawrie, are quoted approvingly in the paper.

"Safety under chloroform can *unquestionably be insured*, but it can only be so by attending to regular natural breathing, and whatever method is employed, no one can deny that it is

the bounden duty of the chloroformist to maintain natural breathing throughout the whole period of administration. To maintain natural breathing requires careful training and considerable experience; but if these conditions be fulfilled it is impossible to produce anything with chloroform but anæsthesia, and the Hyderabad Commission has shown that *anæsthesia alone is entirely free from risk.*"

I want to inquire, On what does this statement of Surgeon Lawrie's rest? According to his own words it rests upon the work of the Hyderabad Commission. What was the work of the Hyderabad Commission? It was a series of experiments upon the lower animals; and here I want to make my first protest. We can never decide by any series of experiments upon the lower animals with positiveness as to the details of the action of a remedy on man. The question whether anæsthetics are safe or are not safe is entirely out and beyond the question of the action of anæsthetics upon the lower animals. It is a question to be settled by the surgeon at the bedside, and, in my opinion, no other evidence is of any value. What is the evidence as to the safety of anæsthetics? You will find in the London *Lancet*, vol. i, 1893, a table in which are recorded 364 deaths occurring from chloroform alone. When to this mass of deaths are added those deaths which have come from ether, from nitrous oxide, from bichloride of ethylene, from bromide of ethyl, and the whole list of anæsthetics, and add also those cases which have been reported in the journals and overlooked by the compiler of the *Lancet's* list, and those cases, by far the most numerous, which have never been reported, we must come to the conclusion that at least one thousand deaths have been produced at the hands of physicians and surgeons by anæsthetics. This statement of Surgeon Lawrie's is to me a most astounding indictment of the whole profession; forsooth, because in their experiments the Hyderabad Commission has reached certain results as to the action of these anæsthetics, we are to believe that anæsthesia is a safe condition, and that these hundreds, and, perchance, thousands of deaths have been the result of ignorance or carelessness. Now, sir, I can believe no such

thing. I state absolutely that experiments upon animals throw no direct light upon such a problem—only a side-light. You might as well say that you cannot purge a man with elaterium because you cannot purge a dog with elaterium. We use animal experimentation, not to determine what a drug does upon man, but to explain the results which we obtain at the bedside and in the clinic.

Now, let us pass on from this point. I want next to consider briefly the immediate cause or way of death. Here, again, I insist that we must refer to clinical results for our knowledge. I find in this paper of the *Lancet* Commission, already referred to, that in the 364 reported cases of death from chloroform, 227 times death came from cardiac failure; in only 60 cases is death noted as having occurred from respiratory failure, and 77 times we are told that the individual died from simultaneous failure of respiration and circulation.

Now, sir, are we to conclude, because certain physiologists allege that they have gotten certain results, that these hundreds of surgeons, men stretching up from the lower ranks to Agnew, Gross, and Simpson, are incapable of deciding whether the pulse or the respiration fails first? I trow not, sir. If the surgical profession be incapable of observing these simple facts, then I am sorry for those who fall into the hands of the surgical profession. It seems to me that the conclusion of the *Lancet* Commission covers the whole ground, that "in the majority of cases the symptoms and phenomena detailed bear out the view that death is due to syncope, in so far as the clinical evidence could decide one way or the other," and I insist that clinical evidence must decide the whole question.

It is to my mind of comparatively little practical importance how ether and chloroform kill the lower animals, nevertheless this question seems to be worth looking at for a few moments. I read in the words of Dr. Hare: "On the other hand, *we do not believe that it is possible in a lower animal (the dog) to cause cardiac death by the freest possible use of chloroform by inhalation without causing primary respiratory arrest*, and respiratory arrest having taken place, the death which follows is partly due to asphyxia and

partly to direct cardiac failure and vasomotor paralysis." As I have said, I consider this point entirely outside of the main drift of my argument, but I state that not only is it possible to have chloroform death from cardiac failure in the lower animals, but Dr. Hare has seen such deaths and has borne testimony to it under circumstances that cannot be gainsaid. I hold in my hand a pamphlet entitled "The Cause of Death from Chloroform," published by Dr. Hare and myself in 1890, and I read there: "We desire to call attention to the fact that of the two tracings here inserted, one was made by injecting chloroform into the jugular vein, and the other was obtained by the inhalation of chloroform. In the second experiment (by inhalation) the breathing continued two minutes after the heart had ceased to act." Not only is this statement made in the text, but you have a reproduction of the tracing, showing that the respiratory movement is quite large and normal in frequency for two minutes after the cessation of the heart's action. Unfortunately, the original tracing, from which this is taken, was not preserved. In preparing for my address on Anæsthesia in Berlin I made a series of new tracings, which were preserved. I would call the attention of the Fellows to these, as they show perfectly clearly the results that I speak of. In No. 5, a weak dog, the respiration continued one hundred seconds after the pulse movement and arterial pressure had fallen to zero, showing that there was no pulse or movement of blood in the open carotid. Experiment No. 9, chloroform in a *healthy* dog, arterial pressure at zero long before respiration had entirely ceased. In experiment No. 13, a healthy dog under ether, respiratory movements of twenty per minute continued sixty seconds after the arterial pressure had fallen to zero. I know that it has been objected that the cessation of movement of blood in the carotid is no proof that the heart has been arrested. Whether or not, under these circumstances, some slight vermicular motion remains in the heart, I do not say, but every surgeon knows that when there is no blood flowing in the carotid artery the heart, for all practical purposes, has ceased action, and if this condition continues the respiratory centres

will cease their function, even if there be no respiratory poison affecting them.

I am happy to see that Professor Lauder Brunton has practically abandoned his case, or at least what I and, I believe, the rest of the world thought was his case. He says that "if we drive chloroform into the trachea, or air heavily charged with chloroform, and maintain artificial respiration, it will be absorbed in sufficient quantity to paralyze the heart." That is the whole controversy—not whether, in dilute form, it paralyzes the heart, but whether it be possible, in the lower animal, to paralyze the heart.

I now come to what is to me a more interesting portion of the subject, because it seems to involve little or no controversy, that is, as to the proper method in which experiments on animals should be applied to this problem. Taking it for granted that the clinical facts vouched for are correct, we try by experiment to understand why these things have happened. What are the phenomena of the circulation affected by chloroform? During chloroform anæsthesia, the arterial pressure begins to fall, and reaches zero if the drug be given in sufficient abundance. The point about which we desire information in order to understand the method of cardiac death is as to the cause of this fall of pressure. There can be but one of two causes for this fall of pressure. Either it is due to the action of the chloroform upon the heart itself, arresting the *vis a tergo*, or it is due to an opening out of the blood paths lessening the *vis a fronte*, or the resistance to the passage of the blood. Let us ask ourselves first whether this drug affects the heart directly. According to the experiments of Ringer, which were quoted by Dr. Hare, the application of chloroform to the frog's heart isolated from the body lessened the output or force. Certainly if you inject chloroform directly into the heart muscle of the mammal it stops the heart instantly, and we find the heart dilated and more or less incapable of responding to stimuli. These facts go to show that this substance depresses the heart. In a very elaborate and interesting series of experiments, of which Dr. Hare acknowledges the force, Dr. McWil-

liams, of England, has shown that during chloroformization there is dilatation of the heart, marked proof that the heart is in a condition of weakness. Now we have recent experiments of Gaskell and Shore, the force of which it has been tried to overcome by pooh-poohing, which seem to prove that the whole fall in the early stages is due to action upon the heart—in other words, the blood paths are not opened out. The experiments were as follows: They took two dogs, side by side, and connected the arterial system of dog A with the arterial system of dog B, and the venous system of dog A with that of the other in such a way that the first dog sent into the brain of the second dog arterial blood which did not pass into the general system of the second dog—in other words, the brain of the second dog was fed with blood from the first dog. It was now found that when the first dog was given chloroform there was a rise in arterial pressure in the second dog—in other words, when the chloroform had access to the brain of the second dog, and no other part, instead of a fall of pressure, there was a rise. There was vasomotor spasm; there was stimulation of the centres in the medulla. Then the experiment was changed so that the chloroform passed to the heart and not to the brain, and now they found a fall of arterial pressure. There is no way of explaining this but by the supposition that chloroform in moderate quantity, whilst it primarily causes lessening of the heart's force, stimulates the vasomotor centres and causes more or less contraction of the vessels. Lauder Brunton published a paper in which he said that these experiments proved nothing. Dr. Hare makes some allusion to them in his paper. I know that this is a complicated and difficult experiment. In weighing such experiments we have to take into account the personal equation of the experimenter, but the fact is that Mr. Gaskell is one of the greatest living physiologists, so far as experimentation on the heart is concerned. His work on the heart has been a new dawn to physiology. Certainly he is a man of great experimental skill, and his name is an evidence that the experiments were properly carried out. Even Lauder Brunton

now admits that the drug acts as a sedative on the heart. That is the point that I want to get at; not whether the respiratory centre of the dog is more susceptible than is the heart, but whether the heart of all animals is depressed by this drug. It might well be that in man the heart is more sensitive than is the respiratory centre, although in the dog the heart is less sensitive than is the respiratory centre.

I wish now to refer to a practical point. The Hyderabad Commission lays immense stress upon the effect of asphyxia upon the heart, and Dr. Hare follows them in this. This is in a certain measure proper. Of course prolonged asphyxia does weaken or depress the heart, but these gentlemen seem to forget that there is another side to the see-saw, that whilst asphyxia oppresses the heart, the failing circulation oppresses the respiratory centres. I see the President shake his head No. In rapid asphyxia there is no sedation of the heart, and it may be that in rapid failure of the heart there is no sedation of the respiratory centre, but in other conditions where the oppression is long drawn out, the organ suffers. It is impossible to make me believe that a heart that is under-fed with oxygen for a time, does not fail. It is equally impossible to believe that a respiratory centre that is under-fed with blood, does not fail. The truth is that the surgeon should watch the respiration, but he should also watch the heart, and when either function fails, measures should be taken accordingly.

I have only a few words more. I want to call the attention of the Fellows of the College to the steadiness of statistics as they grow, as showing the importance of the clinical side of this question. Has it not been twenty-five years since Richardson said that the mortality of chloroform was one in three thousand? Here I have statistics gathered by the industry of Dr. George M. Gould, not of a few inhalations, but of 650,000, and the proportion comes out one in about three thousand. I have long taught from my own results and my reading that chloroform was four or five times as dangerous as ether, and here we have the statistics of Dr. Gould, who, when he started to gather the statistics, was a believer in chloroform as a safe

anæsthetic, covering 1,000,000 inhalations. These statistics show that the proportionate deaths from chloroform and ether are as four to one. Now, sir, unaffected by any of these alleged revolutions we have the same death-rate of anæsthesia running down through the decades. Are we to believe that this tread of death is simply due to the incompetence of the profession, and that chloroform is a safe anæsthetic? No, sir. Anæsthesia will become comparatively safe only when the surgeon recognizes that anæsthesia is an approach to death, and that he ought never give an anæsthetic without recognizing that he is putting the life of the patient in peril and that there is no such thing as a safe anæsthetic.

In conclusion, I want to say a word in regard to a form of using anæsthetics which so far as I know has not been tried in this country, and one which at present bids fair to be of service in at least a few hospitals. This is mixed anæsthesia. I do not mean by mixed anæsthesia the use of any mixture of drugs, but that we begin with one agent and continue with another. It is possible that bromide of ethyl may be found to be a working substance for beginning the anæsthesia, which may then be continued by chloroform or ether, ether of course, in my opinion, if the safety of the patient is consulted. Although bromide of ethyl has been highly lauded, it is not without its dangers. It is excessively prone to undergo decomposition. In a recent journal there is reported a case in which death occurred with symptoms of acute yellow atrophy of the liver, apparently produced by an impure bromide of ethyl. On the other hand, nitrous oxide—ranking entirely apart from the anæsthetics, not an anæsthetic in the proper sense of the term, but simply an agent to shut off oxygen—has been used in St. Bartholomew's Hospital by Hewitt, as a means of commencing the anæsthesia which has been continued by ether. So far the statistics have reached about 13,000 inhalations without any evil symptoms in any case. Nitrous oxide apparently does away with the slowness and disagreeableness of ether. Hewitt has invented an apparatus by means of which

ether is readily thrown into the inhaler as the patient passes from one anæsthesia to the other.

A year or two ago I thought that it might be possible to produce anæsthesia by mixing oxygen with nitrous oxide. Nitrous oxide kills by depriving the tissues of oxygen. I thought that it might be possible to add sufficient oxygen to avoid this and at the same time continue the anæsthesia. There is such a zone and, theoretically, it is possible to do this, but the zone is so narrow that I doubt whether it would be practicable for any length of time. There is always danger that the patient will come back to consciousness or go on to death. The line is too narrow to make it practicable.

DISCUSSION.

Dr. H. A. Hare: I approach the discussion of this subject with some diffidence, for I feel that I have not the power of speech possessed by Dr. Wood. It is only right to call attention to the fact that you can make evidence prove almost anything if you distort it by taking it out piecemeal and not including the collateral evidence. In the beginning of his remarks, Dr. Wood referred to the importance of animal experimentation in regard to man. Later he expresses doubt whether these experiments can be utilized without throwing a great deal of mist about the question, and finally he excludes all such experiments.

Dr. Wood refers to the *Lancet* Collective Investigation, as proving that many deaths had occurred and that chloroform must, therefore, be a dangerous drug. He then reads from Lawrie's statement, quoted in my paper, that "Safety under chloroform can unquestionably be insured," but he fails to state that this statement is qualified by what immediately precedes, viz.: "The Hyderabad Commission's work proves that while Syme's principles are right, there is no such thing as a safe method of chloroform administration," which quite seriously modifies the statement quoted by Dr. Wood.

Dr. Wood asserts that the only basis that Surgeon Lawrie has for his statement is the experiments of the Second Hyderabad Commission, but in many publications and in a letter from Surgeon Lawrie, the latter informs us that he has given chloroform in forty thousand cases without a death. This clinical experience is sufficient to controvert the statement that he has made assertions based upon physiological experiments alone and without clinical support.

Dr. Wood asks if we believe that Gross, Agnew, and others, or such of them as have had chloroform deaths, were careless enough in giving chloroform not to know that the pulse was failing. As I pointed out last spring, I do not believe that these men felt the pulse of any patient dying under their hands. It is the resident physician who feels the pulse and the surgeon is generally informed that the patient is dead a number of seconds after the event has occurred. The surgeon himself does not have the opportunity of examining the pulse to determine whether the heart or respiration stops first.

Dr. Wood says that in the paper we published together in 1890, there is a tracing that shows that respiration continued for two minutes after the heart stopped. In this report that we have made to the Indian Government, there are tracings that show that after the heart has become so feeble that it cannot make the pen move, if you give the dog artificial respiration, he will recover and finally voluntary respiration will return. There are a number of tracings which show this fact. There is no proof that in the experiments of Dr. Wood and myself we gave any one of these dogs a chance to recover, or that we used artificial respiration long enough to give the heart a chance to throw off the poison and again begin beating. I have repeated this experiment so often that I am sure of its correctness. I have brought the pulse wave down to the abscissa line and then resorted to artificial respiration, and in from thirty seconds to two minutes have seen the pulse waves begin to rise and finally become as great as before chloroform was given.

Another important point is that no mist should be thrown around any subject so that only one side is presented. Certainly the impression one would get from hearing Dr. Wood's remarks, is that our report to the Indian Government takes the view that death is due solely to respiratory failure and not to cardiac failure. It is at this point that the quotation of disconnected lines can prove anything. On the third page of this report I quote from the conclusions of the Hyderabad Commission and our own as follows:

"If the chloroform is less diluted the fall is more rapid, but is always gradual, so long as other conditions are maintained; and however concentrated the chloroform may be, it never causes sudden death from stoppage of the heart. The greater the degree of dilution the less rapid is the fall, until a degree of dilution is reached which no longer appreciably lowers the blood-pressure or produces anæsthesia."

To this we add these words:

"With this entire statement our results are practically in accord, but we would like to qualify the words 'however concentrated the chloroform may be, it never causes sudden death from stoppage of the heart,' by saying it never has caused sudden stoppage of the heart in any of our experiments unless respiration ceased primarily. We make this modification because, as we will point out later in this report, we believe circumstances may exist in which the diseased heart may stop suddenly under chloroform."

On page 6, after a statement of the results of the Hyderabad Commission,

we say: "We shall point out later in this report how we differ from this conclusion, for we believe that true fatty heart, plus ventricular engorgement, plus vagal irritation, plus possible valvular disease, and, finally, plus extreme vasomotor relaxation, may result in death in frightened persons. While this is not, scientifically speaking, a cardiac death from chloroform, practically the chloroform is the last straw which upsets the cardiac balance. However, Lawrie and his colleagues recognize this as well as ourselves."

We say that "we believe that in the healthy animal and man we do not have cardiac death." Again, "We believe that this is true of the healthy heart but not of one engorged with blood which has undergone fatty degeneration." In such cases death may take place. Passing on we come to a whole column on page 8 which states distinctly that we believe that death may be due to cardiac failure in addition to respiratory failure, for we state: "Chloroform is capable, therefore, of causing death of the cardiac muscle whenever it comes in contact with it, and that there is no possibility of this arrest being due to vagal irritation is proved by experiments in which vagal section preceded the use of chloroform."

The Hyderabad Commission states: "The truth about the fatty heart appears to be that chloroform *per se* in no way endangers such a heart; but, on the contrary, by lowering the blood-pressure, lessens the work that the heart has to perform, which is a positive advantage. But the mere inhalation of chloroform is only a part of the process of the administration in practice. A patient with an extremely fatty heart may die from the mere exertion of getting upon the operating-table, just as he may die in mounting the steps in front of his own hall-door, or from fright at the mere idea of having chloroform or of undergoing an operation, or during his involuntary struggles. Such patients must inevitably die occasionally during chloroform administration, and would do so even were attar of roses or any other harmless vapor substituted for chloroform."

To this we add: "We agree entirely with this statement; but as chloroform has confessedly some cardiac action and a very positive vasomotor and respiratory effect, the fatal result might be more direct."

In the Summary of this research, Dr. Thornton and myself use these words: "We very positively assert that chloroform practically always kills by failure of respiration when administered by inhalation." We are now speaking *physiologically*, and there is a great difference between a physiological and a clinical statement. If we take a dog and try to discover whether he dies from failure of the respiration or of the circulation, by delicate apparatus we can ascertain which ceases first. If the respirations cease thirty seconds before the heart stops, then *physiologically* that is a respiratory death, but clinically both cardiac and respiratory death ensues. Dr. Wood himself teaches in regard to aconite that it kills by failure of respiration, yet anyone who has seen a case of aconite poisoning knows that while death occurs physiologically from failure of the respiration, yet the heart ceases its

function practically simultaneously within thirty seconds after respiration has ceased."

We say then: "We very positively assert that chloroform practically always kills by failure of respiration when administered by inhalation, provided– and this provision is most important—that the heart of the anæsthetized is healthy and has not been rendered functionally incompetent by fright or violent struggles, or, again, by marked asphyxia. By a healthy heart we mean one which has not undergone true fatty degeneration, or has not so severe a valvular lesion as to make the slightest variation in the even tenor of the circulation fatal.

"As positively as we assert that chloroform kills primarily by respiratory failure, so do we also assert that in excessive dose by inhalation it has a depressant effect on the circulation, which is chiefly due to centric vasomotor depression, with final depression of the cardiac muscle itself. Depression of the cardiac muscle alone is never great enough to cause death when the chloroform is given by inhalation, but we believe that gradual asphyxia, with the direct depression of the circulation, may do much toward producing a fatal result, for vasomotor integrity is almost as necessary to life as an intact cardiac mechanism. This circulatory depression has been considered a safeguard because it was supposed to prevent chloroform going to the vital centres; but in reality it is no safeguard, because profound circulatory depression is as great an evil as respiratory narcosis. That the circulatory depression may be dangerous is not only evident, but it is stated to be so by the second Hyderabad Commission itself at the end of paragraph 8. This circulatory depression may be so profound that recovery is impossible even with the most thorough artificial respiration, a fact stated by the second Hyderabad Commission in paragraph 25, which we quote in this paper. This emphasizes the fact that we cannot afford to totally ignore the effect of chloroform on the circulation."

That we appreciate the difference between physiological and clinical facts is clearly shown. We then add:

"We think that it is just at this point that our research, and every other research on animals, fails, and necessarily fails, to produce a positive reply. The variation in the action of a drug on a diseased individual from its effect on the normal one is notorious, and we have no right to dogmatically assert that there is absolutely no danger of circulatory depression in man, even if we found no evidence of failure in dogs, because there may be many idiosyncrasies or variations, through disease in the human being, which may completely reverse the results of experiments on healthy animals.

"In other words, supposing that the amount of depression from very full doses of chloroform equals 25 units, this amounts to little in the normal heart; but if the heart be depressed 25 additional units by disease, the depression of 50 units may be fatal, particularly if to this 50 is added 25 units more of depression through fright and cardiac engorgement, through

disordered respiration or struggling. That true depression of the heart muscle may take place under chloroform seems to us most undoubted, and we think that the tracings in every research that we have seen support this view. There is always a decrease in cardiac power manifested by the decrease in the force of the individual pulse-beat, and this passes away only if chloroform is removed early enough. We also agree with McWilliams that from the very first inhalation of chloroform there is a constant tendency to cardiac dilatation."

In studying the clinical side of the subject we next propound some questions, among which is: "What is the true cause of death from chloroform?" To this we reply, "that death is always due in the healthy animal to respiratory failure accompanied by circulatory depression, which latter may be severe enough to cause death even if artificial respiration is skilfully used."

It is at this point that I wish to call attention to the results obtained by Dr. Wood, and, I think, obtained erroneously. I do not intend to criticise any physiological process to which he has resorted, but Dr. Wood, when he gives chloroform, may give it in such a way as to frighten the animal. In such cases the dogs struggle violently and cardiac death may occur.

There is one other point to which I should like to refer, and that is that Dr. Wood has shown a tracing of a cardiac death which he states was taken from a weak and sickly dog. This agrees with what we say that in an unhealthy animal it is possible to produce cardiac failure by the use of chloroform. He says that every surgeon knows that when there is no blood passing through the carotid the heart is practically dead, but what proof has Dr. Wood that there is no flow of blood. We have proven that a stream may be flowing, although too feebly to move the salt solution, weighing several ounces, and the mercury in the manometer. This fact was recognized by the Hyderabad Commission when they used Fich's manometer. It requires a pulse-wave of considerable force to register on the mercurial manometer. I have shown this circulation by cutting the opposite carotid after there was no indication of a pulse-wave by the manometer, and the blood trickled out.

Dr. Wood makes a mistake in saying that I quote Ringer. The Hyderabad Commission quotes Ringer.

The point that chloroform depresses the heart when brought in contact with it is of no clinical value. Almost every drug will kill if injected into the heart. I have injected chloroform into the jugular vein without producing as rapid cardiac death as by the injection of quinine. All these powerful drugs will kill if injected, provided they come in contact with the heart in a concentrated form.

The experiment of Gaskell and Shore is a very complicated one. When you are working with such a fine mechanism as that of the vasomotor and nervous systems of the heart, and send the blood from a bull-terrier into a Newfoundland and *vice versa*, you cannot expect that it will affect the vasomotor centres of each animal in the same way. It is the history of trans-

fusion that the passage of blood from one animal to another is accompanied by marked changes in the vasomotor and general circulatory systems. Dr. Wood referred in a very complimentary manner to Gaskell, and I am well acquainted with his work; but, while appreciating the splendid work that he has done, I must deny the value of his experiments in this case, although I do not deny that chloroform does depress the heart. I do not put any weight on the importance of asphyxia in the way that Dr. Wood thinks that I do.

I would finally like to say in order to set myself right that in the healthy man and animal the respiratory function is the first thing affected by chloroform, provided it is given gently and not pushed so as to frighten and cause struggling; after that we get a fall of blood-pressure due to depression of the vasomotor system, and finally, at the end, we have a condition in which the heart-muscle itself becomes depressed. I believe, and in this I agree with the experience of Lawrie from his 40,000 cases and his Hyderabad experiments, that if chloroform is given gently so that the patient passes under it more and more quietly, in any case where death occurs it will be due to respiratory failure, provided that there is no heart disease or other condition interfering with the heart. Chloroform is a dangerous agent in the hands of everyone not most skilled, and it has caused more deaths than ether; but to say that it is such a frightful lethal agent as does Dr. Wood, and that its use should be tabooed, I think is not a correct statement to make. Chloroform does sometimes kill by failure of the heart. There are exceptions to every rule. Some healthy hearts have probably gone down under its use. As soon as respiration is disturbed the chloroform must be stopped, not because the heart is endangered, but because the stormy respirations take into the lungs such unknown quantities that the anæsthetizer has no idea how much chloroform is being given. If chloroform is given gently, so that the respiration is not disordered, then the heart is not disturbed, and the only circulatory change is fall of pressure due to vasomotor depression. To-night, before coming here, I repeated the experiment of giving a dog chloroform gently until respiration ceased, and the heart continued to beat actively for a number of minutes after respiration ceased.

Dr. Wood: I congratulate myself, I congratulate the College of Physicians, and I congratulate all men who come under the weight of the influence of Dr. Hare, that I have drawn him out and brought him to say what he did three minutes ago—that chloroform produces sometimes death by failure of the heart, and that the healthy heart goes down under the influence of chloroform. Having said that, he has admitted all that I have contended for. I think that the long extracts which he read from his work under the statement that I had inferred certain things was precisely like putting up a man of straw to buffet him. I made no allusion to Dr. Hare, save in regard to the action of chloroform in arresting the heart of the animal.

I think that it is hardly worth while to occupy the time of the College in any attempt to follow the remarks of Dr. Hare. The explanation which has

been given by him of heart failure which we reported together as occurring under our observation in a chloroformized dog strikes me as comical. He says that he has had similar tracings, and that he has used artificial respiration and the heart has recovered itself, but we did not give that dog half a chance, and therefore he did not die of heart failure—all this though the dog is dead and buried, and his heart did stop first! He said if we had used artificial respiration the dog would not have died. What of that? the dog is dead, and if the Doctor has seen similar tracings in his Hyderabad experiments, all the more serious for his refusing to allow that a dog may die from chloroform from syncope. If there was anything of value in my Berlin speech, it was in the fact that there first was shown the extreme value of forced artificial respiration in anæsthesia. I showed that you could take a dog whose respiratory centre was practically dead, or you could take a dog whose heart was practically dead and incapable of recovering itself, and by artificial respiration sweep out the chloroform that was depressing the heart and the respiratory centre, and then the natural forces would exert themselves. If we do not sweep out the poison, the dog dies—of the syncope or of the asphyxia, as the case may be, continues until the animal is dead beyond all peradventure of resuscitation. Whereas, if we do sweep out the poison early, the arrest of functional activity which immediately precedes absolute death may be recovered from, and the dog lives—there is a potential death and there is a completed death.

In regard to the remarks about surgeons and resident physicians. That might have done at one time, but it should not do now unless the surgeons are a very careless set of people. The attention of surgeons has been drawn to this point by controversies sweeping over the whole medical world. They have been told over and over, "Your observations are false because you are careless," and still the records are the same. Are surgeons so hopelessly besotted that they cannot learn to be careful enough to tell whether the heart or respiration stops first?

Dr. Hare: Dr. Wood says that he cannot see the use of my remarks because my views agree with his perfectly. I would then ask why he brought up the subject. I have all along said that respiration failed first and the heart afterward in slight measure.

I do not believe that the surgeons are besotted. I believe that the surgeon is so busy attending to the patient that he cannot watch the pulse and respiration also.

Dr. Wood: Dr. Hare asks why I brought up this discussion. It is on account of what I may call the literary style of the Hyderabad Commission report and of this paper. The subject is left in such a condition that no man is quite sure what is meant. Take the one point where Dr. Hare made answer to my remarks about Dr. Lawrie, and which was well taken by Dr. Hare. Lawrie does say, and I had marked it in my copy, that "The Hyderabad Commission's work proves that while Syme's principles are right, there is no

such thing as a safe method of chloroform administration," but going on a few lines farther I find, "Safety under chloroform can unquestionably be insured" I therefore concluded that the first statement was a misprint. How can any man understand an author who says first a thing is not so and then says that it is so: "There is no such thing as a safe method of chloroform administration;" "Safety under chloroform can unquestionably be insured." Again, we are told positively in the paper by Dr. Hare, "We do not believe that it is *possible* to kill a dog by cardiac failure with chloroform," and now Dr. Hare tells us that he does believe that it is possible to do it—which is all I have contended for.

Dr. Richard H. Harte: I have listened with interest to the discussion of these experimental points in regard to anæsthesia. It is a wonder that there are not more deaths than there are from these agents. The general idea is that ether and chloroform can be given with impunity. If they are properly given the mortality from them is reduced to a minimum. In many operating amphitheatres ether is administered by saturating a towel with the drug, and placing it directly over the individual's mouth and nose. This at once causes a sensation of smothering and excites unnecessary struggling, requiring several assistants to hold the patient. If ether is properly administered the individual can be etherized practically without his knowledge. It is the same with chloroform. I have administered chloroform in this city many times, and experience bears out Dr. Hare's statement that the respirations always cease first, although I have never seen a fatal case.

I do not think it wise to employ mixed anæsthesia. If you give ether and chloroform together you do not know what you are giving. If you give them separately you know what you are doing. If ether is properly administered, it can be taken without any effort on the part of the patient in its early administration by slow inhalations, especially through the mouth, as its fumes are irritating to the nasal mucous membrane.

Dr. H. Y. Evans: I had the opportunity of seeing the work of Sir James Y. Simpson when he was at his height. No man was more experienced than he in the use of chloroform. He advised his young men to watch the pupil. He never gave the *rationale*, but insisted that when the pupils dilated the administration of chloroform should be stopped. We now know that the respiratory and cardiac centres are being inhibited when the pupils dilate. This is a warning which I believe precedes any other symptom of respiratory or cardiac arrest. It occurs two or three minutes before any disturbance of respiration is noted.

Dr. G. G. Davis: I wish to say a word in defence of the much-abused resident who is held accountable for these chloroform deaths. If Dr. Hare wishes to prove his point, he must show that it is the resident who is responsible. Abroad, almost every hospital has its official anæsthetizer. Therefore his statement cannot apply to the deaths occurring there. In this city the hospitals of the University of Pennsylvania and I presume the

Jefferson have their official anæsthetizers. Therefore the accidents cannot be attributed to the inexperience of the anæsthetizer except in the other general hospitals.

If I understood Dr. Hare correctly, he said that death under chloroform could occur from cardiac failure under three conditions: first, fright; second, pathological conditions including fatty disease of the heart, and third, at times, in physiological subjects. I do not believe that it is possible to detect and appreciate the *exact* extent of fatty degeneration of the heart. I believe that the degree of fright under which the patient is laboring likewise cannot be accurately estimated, and thirdly, you cannot tell in what proportion of cases the patient is going to die even if physiologically sound. You have then three admittedly uncertain points which no one is capable of determining in chloroform anæsthesia. The lesson that the surgeons draw from these facts is that deaths have been so much more numerous under chloroform than under ether, that the former is an agent for only exceptional use in suitable cases, while the latter is to be preferred for everyday employment.

THE PRESIDENT: Dr. Reichert and myself, in our experiments on snake poisoning, found sudden deaths under chloroform so common that we discontinued its use on account of the expense it entailed to replace the animals. It is not proven that either in animals or man does fright have anything to do with deaths under anæsthesia. Both speakers have alluded to fright as tending to bring about lethal results; but as to this I do not think there is the slightest proof; nor have they given any such.

I have seen many people go under the influence of ether in a state of consternation or of extreme terror, but have myself never been able to perceive that this emotional state in any way added unpleasant or dangerous influences. I am inclined to think that always, despite every precaution, there will be a small percentage of deaths under anæsthetics. I suspect that there is a very small number of individuals who are abnormally susceptible to the dangers of these agents. I know of one case, in an apparently healthy man, where in excellent hands ether has three times produced such results that no physician who has given him ether would be willing to repeat the experiment. I think, even in the laboratory, we now and then find an animal dying under anæsthetics—chloroform especially—in a way which seems to show some extreme susceptibility to its action.

DR. HARE: I can best answer Dr. Mitchell's question by reference to laboratory experiments. Some years ago we had a man connected with the laboratory who was able to supply any number of dogs. We would tell him that we required a certain number of dogs and he would bring them of all kinds, some intelligent dogs and some that had been running wild in the streets. For instance, he would bring an intelligent mongrel shepherd dog. Such dogs nearly always died under anæsthesia unless the agent was administered with great care. On the other hand, the dog that was the scum of the earth took the anæsthetic without any difficulty; he was not frightened

and nearly always survived for the rest of the experiment. I have noticed that if you take a dog and wrap a towel around his head and have him struggling for thirty seconds and then give the anæsthetic, he is more apt to die than the dog to whom you give the anæsthetic gently.

In regard to the evidence from hospitals. I think that every Fellow of the College will admit that while all hospitals may have an anæsthetizer, yet in the large proportion of cases the anæsthetic is administered by the resident. I have seen death occur from chloroform in the hands of one of the greatest surgeons of this country, in a large hospital when the anæsthetic was given by the resident physician. As a general rule, the attention of the surgeon is not called to the condition soon enough for him to determine whether the heart or respiration first fails.

Dr. Wood: I have been anæsthetizing animals since 1867 or 1868. I have never seen any possible relation between fright, intelligence, and anæsthetic death. I should like to see the record of Lawrie's 40,000 inhalations, and I should like to see the record showing that shepherd dogs die in greater proportion than others.

A great deal is said about struggling and there is a certain amount of truth in it. If you tie a dog down and he struggles violently for fifteen or twenty minutes he is exhausted, and, of course, dies more readily. The struggling man or dog is also more apt to die because his fighting leads the anæsthetizer to pour the chloroform on more freely. I have given up the use of chloroform, because with every precaution and the utmost gentleness in administration, I lost many animals.

A German has recently done a thing which is so simple that it is strange that it was not thought of before. He had a patient dying from chloroform. Having inverted the patient without result, he then simply performed insufflation from his own mouth, and in three minutes the danger was over.

Dr. Frank Woodbury: I had the honor to be connected with the clinic of Dr. S. D. Gross, and have heard him state that he had given chloroform upward of 12,000 times and never had had a death. He always had an official anæsthetizer for his clinic. I, however, witnessed some accidents, and the first statement made was that the patient had stopped breathing. Artificial respiration and inversion of the patient was then resorted to, and always with success in the cases that I saw.

The remarks in regard to the condition of the heart I regard as one of the most important contributions to the discussion. Dr. Hare has stated that in cases where the heart was diseased the patient might die from chloroform. Some time ago Dr. Seiler told me that he was making a collection of normal tissue specimens for the microscope, and applied to the hospitals for healthy hearts from patients dying from accident or disease, and that he had to examine upward of one hundred hearts before he could find one that was sufficiently healthy to give him the specimens that he desired.

Dr. S. S. Cohen: I should like to inquire of Dr. Wood and Dr. Hare

whether their observations agree with the careful researches published by Sir B. W. Richardson in 1870, and republished with additions in the *Asclepiad* for 1892, in which he calls attention to certain facts that I have not heard mentioned in this discussion. First, that both animals and man differ individually to a great degree in their susceptibility to chloroform. Among other facts bearing upon this, he states that among dogs placed in the lethal chamber of the Society for the Prevention of Cruelty to Animals some survived and could be resuscitated long after the others died. He likewise calls attention to the great influence of atmospheric conditions, especially moisture and temperature, upon the result of chloroform anæsthesia. The greater the moisture and the lower the temperature, the greater is the likelihood of a fatal result.

Secondly, Dr. Richardson calls attention to the existence of a class of persons whom he terms "morituri." A patient of this class may survive under favorable circumstances, but will inevitably die if anything happens to disturb too far the balance of the nervous functions of organic life. One case is related in which death occurred from fright before enough chloroform had been inhaled to have the slightest effect upon the organs of the patient in any way.

Richardson gives statistics of over 35,000 inhalations of chloroform, with eleven deaths. This he takes to be the normal ratio of fatality, and believes that it must occur under any circumstances, no matter how careful the administrator may be. In the analysis of these cases, he states that neither the apparent condition of robust health of the patient or the pathological conditions present, whether affecting the heart or otherwise, could be determined to have affected the issue in any given case. On the one hand, he refers to the experience of Snow, who had 4000 inhalations in all pathological conditions, including those of the heart, without a death; and on the other hand to a case in which death occurred in a vigorous young person all of whose organs were healthy, and to whom the anæsthetic had been carefully and properly administered. Dr Richardson expressed the belief that the only cardiac condition which could be positively stated to render it likely that the patient would die under chloroform because of that condition, was dilatation of the right heart, giving a theoretical explanation which I shall not repeat, but which all who are working at this subject would do well to read.

Dr. Hare: I have noticed a great difference in the susceptibility of animals both to ether and to chloroform. This varies with the breed of the animal. My impression is that short-haired dogs, similar to the fox-terrier, having a high arterial tension, struggle more, go under chloroform more slowly, and more frequently have accidents than do dogs of the Newfoundland type, which have a comparatively low arterial tension.

I would ask Dr. Wood why he recommends blowing into the lungs, if death occurs so frequently from cardiac failure. Surely that will not start the heart.

Dr. Wood: A high temperature renders the use of chloroform less dangerous. I said in my paper that the reason that Lawrie did not get deaths was because he was in a tropical country, where the vapor was readily carried off.

The reason for blowing into the lungs is to blow the chloroform out.

Dr. Woodbury: The reference to Dr. Richardson calls to my mind a recent contribution as to the causes of death by chloroform. It was held that when chloroform is administered by artificial light the vapor of chloroform undergoes decomposition and an exceedingly poisonous gas is formed which causes a fatal result—if not immediately, in the course of a few hours.

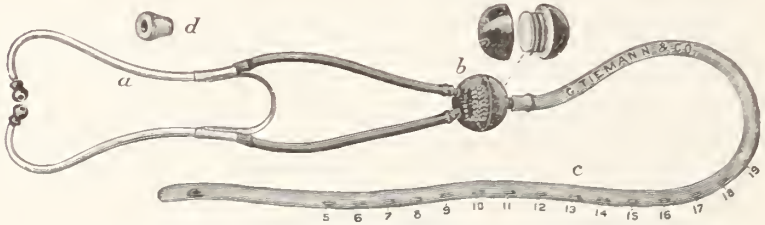
Dr. J. Madison Taylor: I am struck with the large number of personally conducted chloroform anæsthesias made by Surgeon Lawrie, which is stated to be 40,000. This would have kept him extremely busy. A rapid calculation I make would place these at over six a working day for twenty years, or thirteen a day for ten years.

Dr. W. S. Forbes: With regard to idiosyncrasy to chloroform, I cannot speak in regard to any animal below man. In a large experience during the Civil War among soldiers who were selected between the ages of twenty-five to thirty and thirty-five years, I do not recall the occurrence of any striking idiosyncrasy, nor do I remember reading of any. I might also say that I believe that the number of deaths in proportion to the chloroform inhalations was very small. Of course in these cases there was an absence of heart trouble or the men would not have been in the ranks.

EXHIBITION OF AN ŒSOPHAGEAL STETHOSCOPE, WITH REMARKS ON INTRA-THORACIC AUSCULTATION.

By S. SOLIS-COHEN, M.D.,
PROFESSOR OF THERAPEUTICS AND CLINICAL MEDICINE IN THE PHILADELPHIA POLYCLINIC; ONE OF THE PHYSICIANS TO THE PHILADELPHIA HOSPITAL, ETC.

The instrument which I have here is a modification of an instrument suggested by Sir Benjamin Ward Richardson. In the course of that observer's examination of a case of suspected stricture of the œsophagus, in which no obstruction was shown by the "water-gurgle" test and he concluded to pass the stomach-tube, it occurred to him that perhaps if he attached the terminal of his double stethoscope to the stomach-tube he



Œsophageal Stethoscope.

a, Metallic conducting-tubes of "Albion" stethoscope. *b*, Capsule with diaphragm. *c*, Œsophageal tube with indented scale. *d*, Soft rubber caps for ear-pieces.

might notice the presence of friction as the tube passed over the stricture, or the absence of friction and of stricture; in this particular case he found no friction and determined that there was no stricture. It then occurred to him that it might be useful to use this apparatus in listening to the sounds of the heart and possible murmurs in the vessels, as in cases of aneurism, unmodi-

fied by transmission through the chest wall, and that possibly he might be able to hear the sounds of the lungs in the same manner. He also thought that certain sounds generated in the stomach might give information as to the manner in which gastric functions were carried on; while the sounds of the abdominal aorta could be heard through the stomach, unmodified by the pressure which on external auscultation is so often a cause of doubt in interpreting the causation of murmurs.

In attempting to repeat Dr. Richardson's observations, I found that there was occasional danger of the regurgitation of fluid through the tube into the ear-pieces. To obviate this objection I have introduced between the stomach-tube and the ear-tube a rubber capsule containing a diaphragm of gold-beater's skin which serves as a barrier and as a resonator. It can be readily taken apart and cleansed, and new diaphragms can be inserted when needed. I exhibit the improved instrument, as skilfully made for me by Messrs. Tiemann & Co., of New York.

The œsophageal tube is of soft rubber, No. 27 French scale, having a lateral eye near the blunt tip. On the same side as the eye, beginning five inches therefrom and extending to nineteen inches, is a series of indentations, one inch apart, which show how far the eye of the instrument is from the patient's teeth. By experiments outside of the body I have found that the sound heard is transmitted from within a radius of an inch and a half or two inches from the opening in the tube, the sound being most distinct when the eye is in close apposition to the source of sound. I have only used the instrument to listen to the sounds of the heart, not wishing to risk the passage of the tube in cases of thoracic aneurism and not being able to hear through it the lung sounds.

The most striking difference between the sounds of the heart as heard in the ordinary way and the same sounds as heard through this instrument, is the almost uniform quality of the first and second sounds; that is, the sharpness of the second sound as heard by the ordinary method, is wanting in this method. The rhythm is of course the same, the second sound

is the shorter, and the pause is easily appreciated. It is possible to separate the two sounds and hear only the first sound, or only the second sound, or to hear both. At eight to nine inches from the teeth, only the second sound is heard; at ten or eleven inches, both sounds are clearly heard; and at thirteen or fourteen inches only the first sound with, perhaps, an ill-defined second sound. I have not satisfied myself as to my ability to differentiate between the aortic and pulmonary sounds by varying the position of the eye of the tube.

My largest number of observations have been made on persons with healthy hearts, who were under treatment for stomach troubles, and thus accustomed to passage of the œsophageal tube; but I have been able to use the instrument on other patients, with and without cardiac disease; among them two cases in which by the ordinary method of examination I had diagnosticated the existence of mitral obstruction. In one of these two cases mitral obstruction alone was diagnosticated; and in the other, mitral obstruction and regurgitation. In both cases I failed in each of two examinations to hear the obstructive murmur through the œsophageal stethoscope. The regurgitant murmur was heard distinctly, but I could not make out that any part of it was presystolic. I do not know whether there is an acoustic reason for this, or whether it was due to lack of skill with the instrument. I have no doubt of the correctness of the diagnosis.

I have also used the method in one case in which there was doubt as to the coexistence of aortic diastolic and aortic systolic murmurs, but the apparent murmur of regurgitation was not heard through the œsophageal stethoscope, whilst the systolic murmur was heard distinctly and a good closure sound was heard. This I think warranted affirmation of arterial roughening rather than valvular lesion.

While speaking of the use of the tube in doubtful cases of aneurism, Sir B. W. Richardson remarks that the good sense of the physician must determine whether in given instances the method is dangerous, and not to be used.

As to the importance of intra-thoracic auscultation, I can-

not as yet speak positively. I do not know how far it will be applicable in cases of doubtful diagnosis—in others it is not needed—or what its exact limitations are. I consider it, however, one of the methods which we may usefully employ among others in our exact observations, and for this reason I have brought the subject before the College for discussion, in the hope that other Fellows may be led to study it.

It is only right to say that two other communications on the subject of intra-thoracic auscultation, one by a German observer, the other by two English observers, have appeared since Dr. Richardson's publication, and apparently as independent studies. Their conclusions are not cited here, as the subject is still one for individual and unbiased work.

DISCUSSION.

Dr. D. D. Stewart: I think that œsophageal auscultation can rarely be employed except in cases accustomed to the stomach-tube. The first introduction of the tube in any case, but especially in those with cardiac difficulty, is liable to cause so much disturbance of the circulation that it would be impossible to form a correct idea of the usual cardiac condition as regards the normal or adventitious sounds. It is a well-known fact that it is dangerous to introduce a tube into the œsophagus or stomach in cases of suspected aortic aneurism; yet, curiously, if I understand Dr. Cohen aright, Sir B. W. Richardson advises the employment of œsophageal auscultation for diagnostic purposes in these cases.

Dr. Cohen: Dr. Richardson says that the method is not to be employed in any case in which it seems to the observer to involve danger. That covers the whole ground, without specification.

EINHORN'S GASTRODIAPHANE.

Exhibited by S. SOLIS-COHEN, M.D.

I wish to call the attention of the Fellows of the College to the "gastrodiaphane" devised by Dr. Max Einhorn, of New York, which consists of a small electric lamp fastened to a flexible stomach-tube, with cords connecting it with a source of electric power and with a handle to make and break the current. Four storage cells having an electromotive force of 8 volts, or an equivalent number (5 or 6) of freshly charged bichromate primary cells, will operate it. The patient having swallowed two glasses (one pint) of cold water, the end of the tube is lubricated with glycerin and the instrument is then readily swallowed by a person accustomed to swallow the stomach-tube and sometimes by those not accustomed to its use. The examination being made in a dark room, when the current is turned on, the contour and area of the stomach appear very plainly as a luminous red zone on the abdominal wall. The advantage of this method is in determining the presence or absence of thickening in the anterior wall of the stomach, and in determining the exact position of the stomach and the outline of the lesser curvature. It is exceedingly useful in diagnosticating between dilatation of the stomach and the condition termed gastroptosis or sinking of the stomach. I have used the instrument in many normal-sized stomachs and in half a dozen cases in which diagnosis was in doubt. In one case it confirmed the diagnosis of dilatation where the ordinary methods of percussion were unsatisfactory; thus proving the presence of pyloric obstruction, although the cause of the obstruction

was so situated as not to be manifest. In another case, with symptoms of carcinoma, and absence of free HCl, but in which a tumor could not be felt, the thickening of the anterior wall appeared very clearly on the abdomen as a dark patch in the midst of the zone of trans-illumination. In another case in which there was a question whether or not a tumor of the abdominal wall communicated with or was attached to the stomach, we were able to demonstrate that there was no direct connection.

The method has a limited usefulness in diagnosis, its advantage being that it is readily applied and that within its limitations the information it gives is easily interpreted.

DISCUSSION.

Dr. D. D. Stewart: I have used gastrodiaphany for about a year in quite a large number of cases. I have found that the introduction of but one or two glasses of water is not sufficient to render the whole stomach translucent. It permits only the lower part of the stomach, that of the greater curvature, to be seen. Heryng and Reichmann advise the introduction of 1000 to 2000 c.c. of water. In this way the lesser curvature is also outlined. At times you can see a portion of the colon illuminated, the recti muscles, the hypogastric veins, and also the edge of the liver and a portion of the spleen. The great objection is the difficulty of getting the patient to swallow the diaphane. One has first to become used to the tube. For gastrodiaphany to be of any diagnostic use it is necessary to introduce so much water that it causes great discomfort to the patient, and often vomiting, necessitating prompt withdrawal of the instrument.

Dr. Frank Woodbury: This instrument was first presented to the German Practitioners' Society, of New York, five or six years ago, and its use demonstrated. It has not made very great progress since then. If we have to administer a couple of quarts of water before making the examination the diagnosis of dilatation is already made. Where we suspect a growth on the anterior wall of the stomach it might be advantageous to use this instrument. I would suggest instead of glycerin as a lubricant the use of cacao-butter, which is a bland and entirely unobjectionable material.

Dr. Stewart: With reference to lubricants for stomach tubes I would say that I use nothing save water. Glycerin is unnecessary and cacao-butter is not to be thought of.

Dr. Cohen. Einhorn claims, and I agree, that one advantage of his instrument and method over Heryng and Reichmann's lies in the small quantity of water necessary. Einhorn has also the priority in the matter, if that is of any consequence. Glycerin is used to lubricate the glass only, and I find it useful and without drawback of any kind. I have as yet had no trouble such as Dr. Stewart speaks of.

OPERATIONS FOR THE CORRECTION OF INVETERATE PES VARUS.

By G. G. DAVIS, M.D., M.R.C.S.,
SURGEON TO THE GERMAN AND ST. JOSEPH'S HOSPITALS; ASSISTANT SURGEON, ORTHOPÆDIC HOSPITAL.

[Read December 6, 1893.]

In the treatment of the most severe cases of varus the profession, as a rule, admits that operations on the bones themselves are necessary, and that these operations are to be either excision of the astragalus alone, or of a wedge-shaped piece of bone sufficient in size to allow straightening of the foot. The choice of procedures having been narrowed down to these two, I believe the wedge-shaped resection offers so many advantages as to command the preference. Excision of the astragalus alone I believe to be an unnecessary procedure when it proves effectual in removing the deformity; and when operation on the bones is required, the removal of this bone alone will not prove an efficient and satisfactory mode of treatment. I recently had a most satisfactory case, illustrating the two operations.

The patient is a boy, twelve years of age, with marked varus of both feet. He had been unsuccessfully treated in infancy by division of the tendons. His right foot was turned inward until parallel to a line drawn through the two malleoli, and the sole was vertical (see Fig. 1). He walked on the outer border. The left foot was similar to the right, but the deformity was less marked in degree. On account of the right foot showing the more marked deformity, I operated on it by wedge-shaped resection, while for the purpose of making a comparison of the two operations, Dr. Goodman performed an excision of the astragalus on the left.

The plantar fascia of the right foot having been divided subcutaneously, the sole was thoroughly stretched and the incision for the wedge-shaped re-

FIG. 1.

Right foot previous to operation.

section made from just anterior to the external malleolus to the tuberosity of the fifth metatarsal bone. The peroneus longus and brevis were pushed

FIG. 2.

Same foot after correction by wedge-shaped resection.

backward and the peroneus tertius forward. With a chisel and elevator a wedge of bone was then removed through the entire thickness of the tarsus. The anterior line of section passed through the cuboid bone and the posterior portion of the cuneiform bones. The posterior section traversed the anterior

portion of the calcaneum and the neck of the astragalus. This enabled the foot to be brought perfectly straight, with the sole resting flat on the ground (Fig. 2). The foot healed nicely in this position, and never afterward troubled him.

In the other foot the astragalus was removd by a long dorsal incision, and healing also occurred promptly. In this foot, notwithstanding the division of the plantar fascia and anterior and posterior tibial tendons while the sole was brought into a horizontal position, the incurvation and hollowing-out persisted so that when the operation was finished a considerable amount of deformity still remained (Fig. 3).

FIG. 3.

Left foot after excision of the astragalus.

The results which I have seen in these, as well as many other cases, have so firmly convinced me of the superiority of the wedge-shaped resection, when judiciously and skilfully performed, that I think resection of the astragalus alone, as a means of treatment of these most obstinate cases of varus, should be absolutely abandoned. I see no field for it which is not better filled by other measures. Almost all surgeons agree in admitting that the removal of some bony tissue is necessary to correct the malpositions that are sometimes found in cases from the ages of, say, twelve to fifteen or more years. The operations, both of removal of the astragalus and of wedge-

shaped resection, have been largely abused, and the former more than the latter. Removal of the astragalus has obtained

FIG. 4.

Imprint of right foot after wedge-shaped resection.

FIG. 5

Imprint of left foot after excision of the astragalus.

its greatest reputation in cases in which no operation on the bones themselves has been required. Even children a year and a half old, and under, have been subjected to that unnecessary mutilation. Those cases in which it has been

successful in removing the deformity, I am disposed to regard as being curable by tenotomy, with forcible straightening under anæsthesia, and the immediate application of a plaster bandage; this procedure being repeated if necessary.

As an evidence of what can be accomplished by conservative treatment, I show this boy of six years. On admission his feet were as illustrated in Figs.

FIG. 6.

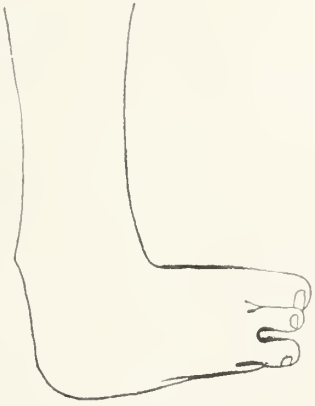


FIG. 7.

Feet of a boy, aged six years, cured without operation on the bones.

6 and 7. They were directed inward toward each other, on a line passing through the malleoli; the soles were vertical, and he walked on the outer

edge of his feet, with his fourth and fifth toes completely doubled under. By means of tenotomy and forcible straightening with the hands under anæsthesia, three times, and then applying plaster bandages, his feet, in the course of three months, were brought perfectly straight, braces being used to prevent relapse. This was a case in which many surgeons would have removed the astragalus, but events proved that it could be cured without it.

I also show you an astragalus removed by another surgeon from a child six years of age. The abnormal bending inward of the neck is well shown. When we examine the bone we find it so soft that I am able to thrust this pin through it in various directions with my fingers. What justification there can be for the excision of the astragalus in such cases I cannot see. The removal of this bone for the correction of pes varus seems to me to be a very illogical procedure. In a foot already shortened on its inner side, the main bone on that side is removed, while the outer side is allowed to remain intact. The deformity is not confined to the astragalus, but the condition and position of the calcaneum and cuboid are likewise altered. The main arch of the foot is composed of two subsidiary arches, an outer and an inner, which are united behind in the calcaneum. The inner arch comprises the calcaneum, astragalus, scaphoid, and cuneiform bones, with the inner three metatarsals. The outer is composed of the calcaneum, cuboid, and outer two metatarsal bones. The outer arch participates in the deformity to an extent as great as, if not greater than does the inner, and, when the astragalus bone of the inner arch is removed the outer arch is still left intact, and in these obstinate cases it is of sufficient strength to still retain the foot in its distorted position of inversion. When one attempts to correct a case that is suitable for operation by removal of the astragalus alone, he at once finds that it is impossible to remove the deformity without proceeding farther; and, first, the external malleolus is chiselled off, then an osteotomy of the calcaneum or cuboid is performed, or finally, a piece of bone is removed, the operation thus practically becoming a wedge-shaped resection. I asked a surgeon once if he could always bring the foot straight by removal of the astragalus, and he said "No." "Then what

do you do?" said I, and he answered: "Go on removing bone until I can bring it straight"—and if the proper cases present themselves to the surgeon that is just what he must do. The greatest objection to the removal of the astragalus alone is that it is unnecessary in the less severe cases, and in the bad ones it fails to remove the deformity. The foot is likely to remain inverted and to retain the exaggerated hollow of the sole and the projection of the dorsum which it possessed before the operation, and which is shown in Figs. 3 and 5. As time passes, the condition at the ankle will probably be satisfactory, but the inversion of the foot, if not carefully guarded against, may become worse, or at least fail to improve, and the patient still be badly crippled. I also believe that after excision of the astragalus at times—particularly if any marked motion remains at the ankle—control over the foot is more likely to be partially lost, causing the toes to drop and the foot to invert in the same manner as is seen in cases of paralysis of the extensors in anterior polio-myelitis. It is objected that wedge-shaped resection mutilates the foot too much, but this is not so, because it is only performed in the already extremely deformed cases. In this boy, the foot on which the operation of wedge-shaped resection was performed, although originally worse than the other, is not only a nice, straight, flat foot, but it is absolutely greater in length than the one in which the astragalus only was excised. The difference in length in favor of the wedge-shaped excision is fully three-quarters of an inch. In fact, the deformity remaining in the foot in which the astragalus has been removed is so marked even now, two years after the operation, that it is necessary for the boy to wear a brace to keep the foot from again becoming worse, as without it the foot turns in and the boy walks on its edge, and, if he persisted, would soon have it completely turned over so as to walk on its dorsum. The condition is so dubious that if not carefully watched it may even yet require an osteotomy or resection of bone to such an extent as may be sufficient to make it a respectable and reliable organ of locomotion.

The tendency among orthopædic surgeons in the treatment

of marked cases of club-foot is toward conservatism and the avoidance of operative proceedures on the bones; but the plaster cast that I now show you (Fig. 8) of my first case of wedge-

FIG. 8.

Club-foot in a man aged twenty-one years.

shaped resection, from a man aged twenty-one years, will, I am sure, convince you that such enormous and inveterate distortions and deformities cannot be satisfactorily treated by mild measures alone.

Mr. Brodhurst, in his recent brochure on the treatment of club-foot, strongly condemns tarsotomies and tarsectomies. The utmost limit to which he is willing to go is shown by the following quotation: "There are, however, certain cases, and among thousands perhaps as many as could be counted on the fingers of one hand, where it would be permissible to remove the astragalus." He therefore admits that operations on the bones are sometimes justifiable, but I cannot agree with him, for the reasons given, in thinking that the operation chosen should be the removal of the astragalus alone. He quotes Mr. Walsham, as follows: "I have always taught that, though I firmly believe these intractable grades of the deformity can only be cured by tarsectomy, at the best it is but a bad job." With this I must dissent, and I do not believe that any fair-minded surgeon would characterize the results in the cases

which I have shown as being, to use Mr. Walsham's expression, "a bad job." (Fig. 9.)

Dr. Ogston is also quoted to the effect that "Tarsotomy by subcutaneous puncture and chain-saw was performed five times,

FIG. 9.

Same after wedge-shaped resection.

but is now given up. Excision of a wedge of the tarsus was performed three times, and, though it looked pretty, was not useful."

If reducing very greatly the amount of deformity and enabling the patient to conceal the remainder, and giving him a painless and satisfactory foot on which he can walk without the aid of braces is not a "useful" procedure, then I would like to know what constitutes usefulness. If the operations quoted above were all performed on a single case it would be a hopeful man indeed who would expect a "useful" foot to result, and if Mr. Brodhurst forms his opinion of the value of the operation in properly selected cases on such procedures as the above, and the method as witnessed by Senn, and described by him in his *Four Months Among the Surgeons of Europe*, then we can readily account for his bad opinion of it. As for myself, I respectfully decline to accept such chain-saw surgery as being the criterion by which to judge of the value of this operation.

DISCUSSION.

Dr. A. Hewson: I would refer to a case operated on at the Jefferson College Hospital in a man twenty years of age, who walked entirely upon the outer portion of the foot. In that instance the astragalus was removed and a section of one of the tarsal bones made. The result was most happy, and the man was able to walk on the sole of the foot without any apparatus. The deformity was exactly as represented on the board.

Dr. T. S. K. Morton: I wish to testify to the value of bone excision. I have come to think that it is *the* operation where there is distortion and actual displacement of the astragalus. I have seen cases equally as bad as the one pictured by Dr. Davis, with the patient walking on the dorsum of the foot, where, by excision of the astragalus and portions of such other bones as interfered with the correction of the deformity, the most satisfactory results were obtained. I have thus operated a number of times, and have seen many more in the hands of Dr. Thomas G. Morton, and that this is the only available procedure in these inveterate cases I have not the slightest doubt.

TWO CÆSAREAN SECTIONS AND THREE SYMPHYSIOTOMIES:

A Year's Work in the Surgical Treatment of Insuperably Obstructed Labor.

By BARTON COOKE HIRST, M.D.

[Read December 6, 1893.]

An insuperably obstructed labor is, I think, as dangerous a condition as can be found in medicine or surgery. It is surely fatal for mother and child unless delivery is effected by a difficult operation, and yet the serious nature of the case is often unsuspected. A grave disease or a bad injury is not commonly overlooked, but an insurmountable obstruction in labor is very likely indeed to remain undetected until the mother and child are past aid. The general practitioner trusts too long to Nature, as a rule, and when he discovers that natural forces are insufficient, he tries forceps and version, procedures that have stood him in good stead before. It is often only when these fail that he realizes the threatening character of the case and calls upon the expert, whose efforts are handicapped by the original attendant's delay. Fortunately, the five cases about to be reported do not strikingly illustrate this fact, but they do show some of the commonest obstructions to be anticipated in labor, and they may, perhaps, serve as a warning to the obstetrician to be on his guard against too long a delay in similar cases. They certainly furnish a strong argument in favor of careful pelvic exploration, including pelvimetry, in pregnant and parturient women.

Case I. *Primipara in labor many hours, with pelvis choked by a fibroid attached to the cervix; Porro operation; recovery.*—This case has been reported[1] by my friend Dr. Guy Hinsdale, under whose care the patient was. The child was, unfortunately, dead at the time of operation. The woman had a shock temperature and pulse—97° and 140—when the operation was begun. She reacted immediately afterward and made a good recovery. One of the chief elements of success, I think, was the rapidity of the operation—less than half an hour—which was obtained partly by certain steps in the technique which I strongly favor. The abdominal incision was long enough to turn the uterus out of the abdominal cavity before it was incised. The intestines were at once covered with sterilized towels placed in the abdominal cavity. The cervix was transfixed with pins and secured by a round rubber band, which was tied in a single knot and the ends then clipped by a very short hæmostat that I have for the purpose. I consider this much superior to the wire nœud. The extra-peritoneal treatment of the stump is not an ideal method, but it is quick and easy, and is, therefore, to be preferred when every moment saved is of importance to the woman.

Case II. *Face presentation with chin posterior and gigantic overgrowth of the fœtus (fourteen pounds); Porro operation; recovery.*—This woman had had several children before without special difficulty. She had been in labor a comparatively short time before she was brought to the Maternity Hospital by Drs. McFadden and Hammond. Efforts had been made to deliver with forceps, but they had failed. When I first saw the woman she was somewhat shocked; the uterus was tetanically contracted; the contraction ring was not far from the navel. The vulva presented the most remarkable appearance I have ever seen. The labia were so œdematous that they projected three or four inches from the body; they were hard, red in color, and in spots contused and abraded. The vagina also showed abrasions. The child's face was tightly wedged in the pelvis, with the chin posterior. I was unable by any plan to budge the face in any direction. As soon as the woman could be prepared a Porro-Cæsarean section was performed. It required all the force I was capable of to dislodge the face from above. The child was dead and its body, as well as the uterus, fairly stank. My hands were infected in the operation, and I was disabled for two weeks. The nurse in attendance on the case afterward had a similar mishap.

Someone inexperienced in this complication of labor—face presentation, chin posterior, gigantic overgrowth of the fœtus—might inquire why craniotomy was not attempted. This operation received careful consideration, but was discarded for the following reasons: It was not certain that the child was dead; an attempt to extract the child through such a vagina

[1] Medical News, 1893.

and vulva as this woman had would have been followed by serious injury, sloughs, and infection; as it was, the left labium sloughed off after the operation; finally, I am convinced that an attempt to deliver by craniotomy would have failed, and that I would have been compelled eventually to perform Cæsarean section with much diminished prospects of success. A medical friend had this experience last summer in a similar case, and I have the specimens from another fatal case in my collection in the University.

Case III. *Primipara in labor forty-eight hours; rhachitic pelvis; symphysiotomy and delivery with forceps; recovery.*—This case has been previously reported.[1] I thought it at the time the first in America, but soon discovered that I was anticipated two days by Dr. Jewett, of Brooklyn. I am now told by Dr. Harris that I was preceded six months by an operator in the Southern States, who is shortly to report his case, the first in America. My patient and her baby were sent back to Germany in good condition.

Case IV. *Rhachitic dwarf, primipara, aged thirty-four years, diagonal conjugate of 9 cm., delivered by prematurely induced labor, artificial dilatation of the cervical canal, symphysiotomy, and version.*—This case has also been previously reported.[2] The delivery was effected with remarkable ease and rapidity. The mother had an afebrile convalescence, and the child, seen last at six months of age, was in a fine condition. A very valuable aid in this case was the slight diminution of the head diameters, the child being between two and three weeks premature, and the thorough dilatation of the cervical canal by a Barnes bag twice as large as the largest sold in the shops, which I had made to order.

Case V. *A rhachitic dwarf, a primipara, four feet one inch tall; diagonal conjugate, 9 cm.; symphysiotomy and version; recovery.*—This case, not hitherto reported, is, on the whole, the most instructive I ever attended. My attention was called to the woman when I first assumed charge of the Maternity Hospital this autumn. It was obvious at a glance that her pelvis was deformed. The diminutive size and the peculiar waddle by which she progressed indicated a serious pelvic deformity. I at once measured the pelvis, and found a diagonal conjugate of 9 cm. I was surprised to find it so long. I repeated the measurement several times, always with the same result. Two of my medical friends found, independently, the same measurement. It seemed a case, therefore, for symphysiotomy. I had delivered a rhachitic dwarf with the same measurement both antero-posteriorly and transversely a few months before without difficulty, and there are many cases on record delivered successfully by symphysiotomy with as small or smaller

[1] Medical News. [2] Ibid.

measurements. I had my misgivings, however, and would have preferred Cæsarean section, but I felt constrained to resort to symphysiotomy on account of its much lower mortality, and in view of its probable success. Labor was induced, the cervical canal artificially dilated, the symphysis cut, and forceps tried. This failed to engage the head. Version was performed and the child extracted up to the shoulders. So much force was required to extract these and the head that I broke a clavicle and the child's neck. I felt convinced by this experience that the true conjugate was smaller than one would ordinarily be justified in assuming with a diagonal conjugate of 9 cm.

To determine this point, and to provide for my use a more accurate system of pelvimetry than Baudelocque's plan, I had this pelvimeter constructed. I found by it that the woman had a true conjugate of less than 6 cm.

To measure the conjugate with this instrument the woman is placed in the lithotomy position, with the buttocks projecting well over the edge of the table. A mark with a lead-pencil is made on the skin over the symphysis one-eighth of an inch below the upper edge. Two fingers of the left hand are inserted in the vagina to find the promontory of the sacrum, as in Baudelocque's method. Tip B of the pelvimeter is then placed firmly upon the middle line of the promontory, and an assistant adjusts tip A directly over the mark made upon the skin covering the symphysis. This arm is then screwed fast, the pelvimeter withrawn, and the distance between the tips measured with a tape measure. This is the true conjugate plus the thickness of the upper portion of the symphysis. The usual thickness of the symphysis at its upper part I have found from the measurement of fifty-five pelves to be 1 cm. Twenty-six pelves had this measurement; thirteen had $1\frac{1}{2}$; nine, $1\frac{1}{4}$; four, $1\frac{3}{4}$; and only three measured 2 cm. One of these was a high-grade rhachitic pelvis, one was of the masculine type, and the third was a justo-major pelvis. I think my case demonstrates forcibly the superior accuracy of this method of finding the true conjugate. The idea, it will be recognized, is borrowed from Wellenbergh and his recent follower, Skutsch, but this pelvimeter answers the purpose much better, I think, than any hitherto constructed.

It is needless to argue for greater accuracy in pelvimetry if

we are to base operative procedures on the difference of less than $\frac{1}{2}$ cm. in the conjugate: the necessity for it is apparent. That the method at present in widest use, Baudelocque's plan of taking the diagonal conjugate, is untrustworthy is well known to all experienced in pelvimetry. Mine is by no means the only case in which there has been a difference of 3 cm. or more between the true and diagonal diameters, instead of the $1\frac{3}{4}$ cm. usually allowed. Litzmann measured a pelvis in which the difference between the true and diagonal conjugates was 2.8 cm. Pershing found one in Philadelphia in which the difference was 3.6 cm., by actual measurement of the dried specimen. It is easy to lay down rules to avoid this error, based on the study of dried specimens, but it is practically impossible to put them to use in the living female. The height of the symphysis is readily found, but an allowance for variations in this respect avoids few errors. It is the variations of the conjugato-symphysial angle that count, and there is no way of accurately measuring them.

DISCUSSION.

Dr. Robert P. Harris: It was supposed until recently that the first symphysiotomy performed in the United States was that of Dr. Jewett, of Brooklyn, September 30, 1892, but the first operation, as Dr. Hirst has said, was performed in Alabama, on March 12, 1892. The case has not been reported, but soon will be. The woman and child, the latter now twenty-one months old, are living. There have been in the United States, counting this case, thirty-one symphysiotomies in twenty-one months. There have also been three in Canada and two in Brazil. The first Canadian operation was performed in Montreal, on December 5, 1892. We have had in all, this side of the ocean, thirty-six symphysiotomies, with four deaths among the women and nine among the children. The most encouraging feature about the operation is the fact that but one death has followed from the last fifteen operations, and there is a possibility of saving a much larger proportion of cases than has been done. One patient died of shock and exhaustion twelve hours after her admission to hospital, with a pulse of 150. A second case was supposed to have had septicæmic inflammation set up in consequence of prior treatment. A third died from double pneumonia set up by exposure on her way to the hospital after imbibing pretty freely of whiskey. The fourth case died on the eleventh day from sepsis in the wound itself.

NOTE ON THE EFFECT OF MASSAGE ON THE BLOOD-COUNT.

By JOHN K. MITCHELL, M.D.

[Read December 6, 1893.]

Recently in studying the results of massage it has occurred to me to examine the blood before and after its use. I have thus far had only a small number of cases, but as they all point in the same direction I think it well to mention the observation. In some six cases examined I have found that massage increased enormously the number of red corpuscles.

The first case was one of extreme anæmia, where the patient had probably suffered from very chronic lead-poisoning. Examination before massage showed 3,725,000 red corpuscles and only a little over 30 per cent. of hæmoglobin. The examination was repeated by Dr. Burr on another occasion, with practically the same result. The patient was then ordered to have abdominal massage only, with the expectation that some result might be gotten through massage of the spleen. This was continued for twenty-five minutes, and the blood-count made immediately afterward showed 4,500,000 red corpuscles. The next day there were 4,000,000. He then had general massage lasting one hour, and the blood-count made ten minutes later showed 6,500,000.

The second case was one of malarial cachexia with choreic spasm of the face in a woman forty years of age. There were a little over 4,700,000 red corpuscles, with 70 per cent. of hæmoglobin. The proportion of white to red corpuscles was 1 : 120. After one hour of massage, she had just under 7,000,000 corpuscles, and the ratio of the white had increased to 1 : 38.

The third case was a perfectly healthy man in good condition. His count was 5,675,000 and hæmoglobin 110 per cent.

After massage the count was made by Dr. Burr, who found 7,900,000, and the hæmoglobin increased to 120 per cent. At another examination the red corpuscles were found increased to 8,000,000.

The fourth case was one of suspected or doubtful locomotor ataxia which had been at rest for several weeks with daily massage and a great deal of food. The first count gave 6,500,000 red corpuscles. The second count was 7,325,000. After massage it was 7,340,000.

In the fifth case, a patient recovering from a transverse myelitis, who had been masséed for some months, the count without massage was not quite 7,000,000, and hæmoglobin 90 per cent., and after massage 8,100,000, hæmoglobin 100 per cent. The examination was repeated by Dr. Burr with the same result.

I hope subsequently to go more thoroughly into this subject and examine a larger number of cases. I mention my results now because they seem opposed to the usual averages given of the number of red corpuscles, and because they show another reason which has not been before mentioned for the usefulness of massage. Wherever the corpuscles are stored or manufactured, they are brought out in large numbers by this process. In two cases the white corpuscles were increased, and in the case where they were most increased they were already slightly in excess. When one recalls the appearance of a bloodvessel under the microscope and how the white corpuscles hug the walls of the vessel, it does not seem extraordinary that they should be pushed out more rapidly into the circulation by the squeezing and pumping of the massage manipulation. The discovery, if verified by further observations, will have important physiological and clinical bearings.

DISCUSSION.

DR. H. A. HARE: The subject brought forward by Dr. Mitchell is of very great interest, and the suggestions which he has made open up a field of original research in which many might be active for a long period of time without exhausting it.

As is well known, a great deal of work has been done with regard to the relative number of corpuscles under different conditions of the body. Ten years ago, when I was very much interested in this question, I found by practical experience and from the literature of the subject, that very extraordinary variations in the relative number of the white to red corpuscles take place before and after meals, and that this change was so frequently seen as to seriously vitiate all blood-counts not made with the greatest possible care. Early in the morning the proportion might be 1 : 250, and after a good meal this relation would be changed to the extent of 50 per cent.

Dr. B. Alexander Randall: It would be interesting to study whether or not there is such imbibition of serum by the manipulated tissues as to lessen the proportion of serum in the blood current and thus apparently increase the number of corpuscles.

Dr. Joseph Leidy, Jr.: It may be of interest to refer to the change that takes place in the number of corpuscles and the hæmoglobin in the condition known as shock. Some years ago I saw the statement that during shock there was a decrease in the number of corpuscles and a diminution of hæmoglobin. At that time I was interested in the investigation of the organisms found in malaria, and in the course of my examinations I examined the blood in a number of cases of shock, and found that there was a decided decrease in the number of red corpuscles in simple shock—that is, those in which there had been no loss of blood, with a reduction also in the percentage of hæmoglobin. The explanation is a mechanical one: During inaction, or while the individual is at rest, there is less blood in the superficial circulation than is found during exercise. It is naturally what one would expect under a stimulus such as massage.

Dr. A. C. Abbott: In connection with this report of Dr. Mitchell it might be well to call attention to Dr. Thayer's observations upon the effect of cold baths on the elements of the circulating blood. The details of these observations have escaped me, but the main points were that after the cold-bath treatment of typhoid-fever patients there was suddenly a conspicuous increase in the number of leucocytes in the blood, and the same result was seen to follow cold bathing in normal individuals.

Dr. Mitchell: The fallacies to which Dr. Hare has referred have frequently been brought to my attention in the examination of the blood. I never make an examination of the blood without noting the time of the last meal. In the cases to which I have referred the examinations have all been made at about the same hour of the day and at about the same period after eating, in order to obviate, as far as possible, the influences of exercise and food.

REMARKS UPON THE COMPARATIVE DIFFERENCE BETWEEN CERTAIN WOUNDS INFLICTED BY THE PROJECTILES OF THE LARGE AND SMALL CALIBRE HAND-WEAPONS.

By CAPTAIN LOUIS A. La GARDE,
U. S. ARMY.

[Read May 3, 1893, by Dr. Guy Hinsdale.]

The subject of gunshot wounds has received a new impetus, for the military surgeon at least. It may be said that we have arrived at another epoch in the development of the military rifle. Since the publication of a pamphlet by Prof. Hebler, a German artillery scientist, in 1888, advocating the small-bore rifle, the foreign governments, one after another, have adopted the small-bore weapon.

The ballistic feats of the small-bore guns have been lauded in the daily press and medical journals to such an extent that the velocity and penetration of their projectiles seem well-nigh incredulous. That the new weapon is superior to the rifles of ten years ago propelling leaden projectiles there is no doubt. Chauvel and Nimier[1] state, as a result of extensive experiments with the Lebel rifle, the portable gun of the French army, that its projectile has velocity and penetration sufficient to make it redoubtable for the human body at 2775 yards. In a recent lecture by Dr. Johann Habart, Royal and Imperial Regimental Surgeon of the Austrian Army, we read that the projectile of the new Mauser rifle—the hand-weapon of the German army—which corresponds very nearly to the Lebel of the French, can penetrate with great facility all parts

[1] Traité pratique de Chirurgie d'Armée, 1890.

of the human body of small density and resistance at even a distance of 5000 steps, and he cites a case in proof of this assertion.

Among the many advantages of a small-bore gun pointed out by Hebler the following may be especially mentioned:

1. Lighter ammunition.
2. Flatter trajectory and greater dangerous space.
3. Less deviation by wind.
4. Less recoil.
5. Greater penetration.
6. Greater accuracy.
7. The wound produced, while being sufficient to disable, is much more humane.

The United States Government is the last among the great nations to change its armament to the new order of things. Although we have every reason to believe that the small-bore rifle will be adopted for our army, still the change has not yet been made.[1] This tardiness is possibly due to the fact that we are not in imminent danger of war, and, further, to the reluctanee which we should naturally find in adopting a gun of foreign make. All the improvements in portable hand-weapons of late years have found their origin in Europe. The well-known genius of the American seems to have been directed in other channels. The Magazine Board, which was ordered two years ago to examine into the merits of the different weapons, has lately recommended the adoption of the Krag-Jorgensen rifle, but the recommendation has not received final approval. We have, however, adopted a projectile which is to be used by the Krag-Jorgensen or whatever gun we may hereafter adopt.

This projectile is now manufactured by the Ordnance Department at Frankford Arsenal, and it eorresponds in resistance to the projeetiles of the small-bore rifles of the foreign powers.

For the purpose of ascertaining the effects of this projectile

[1] The U. S. Government has, since the reading of these notes, adopted the Krag-Jorgensen rifle. The regular army and the national guard will be provided with the new armament in the course of one or two years.

our ordnance officers have manufactured the Experimental Springfield Rifle of 0.30 calibre. This weapon, with the projectile referred to, possesses for all practical purposes ballistic qualities equal to those of the new Mauser, the Lebel, Maennlicher, etc.

The two weapons which especially concern us this evening are the 0.45 calibre Springfield rifle and the 0.30 calibre Experimental Springfield rifle.

The following is a description of the more important ballistic properties of these weapons, as kindly furnished by authority of the Chief of Ordnance, U. S. Army:

"The Springfield rifle, which has formed the armament o our foot troops since 1874, is a 0.45 calibre gun, the projectile of which has an initial velocity of 1301 f. s. Its projectile is made of compressed lead, cylindro-conoidal, cannelured and lubricated; weighing 500 grains; impelled by 70 grains of black rifle powder.

"The Experimental Springfield rifle is a 0.30 calibre gun, the projectile of which has an initial velocity of 2000 f. s. Its projectile weighs 220 grains, is made of a German-silver jacket filled with a lead core, and it is neither cannelured nor lubricated. It is impelled by 36 grains of smokeless powder.

"The velocity and energy of the projectiles of the two weapons at different ranges are as follows:

VELOCITY OF THE PROJECTILES OF THE TWO GUNS.

Name and calibre of weapon.	Initial velocity, f. s.	500 yds	1000 yds	1500 yds	2000 yds
Springfield calibre, 0.45	1301	873	676	531	429
Experimental Springfield calibre, 0.30	2000	1103	804	627	495

ENERGY OF THE PROJECTILES IN FOOT-POUNDS.

Name and calibre of weapon.	Initial velocity.	Weight, grains.	Muzzle.	500 yds	1000 yds	1500 yds	2000 yds
Springfield calibre, 0.45 . . .	1301	500	1879	846	507	313	204
Experim'l Springfield calibre, 0.30	2000	220	1954	594	315	192	120

"It will be seen by the first table that the initial velocity of the small-calibre projectile is far greater than that of the large-calibre and that the striking velocity at all the ranges is greater.

"The penetrations of bullets are proportional to the squares of their striking velocities; for equal velocities the penetration is proportional to the density of section. The form of the bullet and its power to resist deformation are also concerned in the penetration."

The superiority in the motion of translation, as well as the greater striking velocity of the 0.30 calibre projectile, is due in great part to the primary impulse, which is greater, to the lengthening of the bullet, and to the reduction in diameter of the missile, which necessarily encounters less resistance.

The projectiles of rifled guns are given two motions as they are impelled from the piece: First, a motion of translation; second, a rotary motion upon their long axis produced by the rifling in the barrel. In the 0.45 calibre gun under consideration the rifling describes one turn in every twenty-two inches. This rotation is sufficient to keep the projectile, which is larger and shorter, cone-end forward; but in the reduction of the calibre, as for instance in the 0.30 calibre gun under consideration, it was found necessary to describe as much as one turn in every nine inches to keep the long thin missile cone-end first. The velocity of rotation for the Springfield rifle is about 800 turns per second, whilst the projectile of the Experimental Springfield rifle describes as many as 2000 turns in the same space of time. This velocity of rotation is sustained, as a rule, without sensible loss at all the ranges, and it exercises a great influence upon the flight of the missile and the correctness of fire.

Having been ordered by the Secretary of War, July 20, 1892, to conduct certain experiments with the projectile of the Experimental Springfield rifle which would show the destructive effects of this missile upon the human body, I proceeded to Frankford Arsenal, the place designated for the experimental work. Through the kind offices of one of your officers I was

enabled to obtain ten cadavers, which were delivered at the Arsenal from time to time, and with the able co-operation of the Commanding Officer at Frankford Arsenal, and his very obliging assistants, I commenced the work of shooting on the 10th day of January, 1893, and fired the last shot on the 25th day of March.

Having been designated by the Secretary of War, about two years ago, to preside over the Medical Section of the War Department's exhibits at the World's Columbian Exposition, I conceived the idea at the onset of the work at Frankford Arsenal of preserving the specimens of bone lesions and the missiles producing them, as a collection to be exhibited among the articles to be displayed from the Army Medical Museum. The great majority of the specimens are now before you. For their preparation the credit is due to the Curator of your Museum, Dr. Guy Hinsdale. Aside from the tedious work of preparing and mounting each specimen, I am deeply indebted to him for invaluable assistance in securing accurate information of the destructive effects by the missiles in the bony structures especially.

Lieutenant J. Walker Benet, Ordnance Department, U. S. Army, who had charge of the ballistics during all of the work, describes the method of computing penetrations and velocities with reduced charges, as well as the method of collecting and identifying the projectiles, as follows:

"A LeBoulangé chronograph was used, the distance between the first and second targets being 100 feet, and the first target being 3 feet from the muzzle: thus the velocity recorded was that at 53 feet from the muzzle. The remaining velocities of both bullets used (500 grains, 0.45 calibre; and 220 grains, 0.30 calibre) were computed for the desired ranges by the methods given in Ingalls' *Exterior Ballistics*. To find the penetration at a given range, say 1000 yards, the procedure was as follows: The remaining velocity at this range, computed as above, was found to be 676 f. s. for the 500-grain bullet. The charge of powder was successively reduced until a charge was found which gave, at 53 feet from the muzzle, a velocity of 676 f. s.

Cartridges were made up with this charge, and the cadaver to be fired at was placed 53 feet from the muzzle. The striking velocity of the bullet being the same as that of a bullet fired with the full charge and striking an object at a 1000 yards distance, the penetration also would be the same. At the ranges (1500 and 2000 yards) it was found necessary, in order to record the low velocities obtained, to reduce the distance between targets to fifty feet, which caused the velocity to be determined at 28 feet from the muzzle. At these ranges, therefore, the cadavers were placed 28 feet from the muzzle. A tackle was provided for traversing the cadavers and bringing the portion of the body to be fired at into proper position. Barrels, filled with sawdust, were placed behind the cadaver experimented with, to catch the bullets and preserve them from deformation other than that received in their passage through the body. Each bullet was marked on its base with a number or letter for the purpose of identification, and after firing at each range the bullets were collected."

In order to make the destructive effects of the small-bore projectile more apparent, I followed the plan of—

1. Noting the effects of the projectile of the 0.45 calibre Springfield rifle upon different parts of the human body at various ranges.

2. Noting the effects of the projectile of the 0.30 calibre Experimental Springfield rifle upon similar parts of the human body, or parts offering about the same resistance, at similar ranges.

Impact.—The shock, as shown by the oscillation of the limb, when a resistant bone was hit was always greater with the leaden bullet of 0.45 calibre than with that of the smaller-bore gun; on the other hand, the difference was reduced to a minimum when the soft parts alone were hit. It often happened that the occurrence of a fracture by the smaller projectile was determined only after a careful examination of the wound. This difference in shock was noted at all the ranges, but especially so after the 500-yards range.

The minimum amount of shock for the smaller projectile

depends upon its superior penetration, which, as stated already, is due to its smaller calibre, greater velocity, and last, but not least, to the fact that it preserves its shape unaltered. The fact that the calibre alone of the 0.45 calibre Springfield was greater would be cause sufficient for a greater amount of shock upon impact; since, however, deformation is the first thing that happens when the leaden bullet collides with a resistant bone, part of the energy is consumed in the flattening and the rest is conveyed to the part struck in the form of shock. The difference in the amount of shock is at once suggested in Table No. 2, already cited. It will be seen that the work in foot-pounds for all the ranges is greater with the projectile of the 0.45 calibre Springfield rifle. Delorme and Chavasse, who have experimented a great deal with the 8 mm. gun of the French army, while commenting upon similar experience, express the opinion that the general as well as the local shock will be less with the new projectiles having metallic mantles.

Explosive Effects.—The explosive effects caused by the projectile of the Springfield rifle were noticed up to 200, and in some instances to 250 yards, whilst the explosive effects of the projectile of the Experimental Springfield rifle extended in some instances as far as the 350-yards range.

The term "explosive effects" is somewhat confusing, as it is apt to convey the idea that the wound was caused by an explosive bullet. It is a term that doubtless owes its origin to the similarity in appearance of the two wounds. When we say that a wound shows explosive effects we mean that it appears as though it had been caused by an explosive bullet. There are no special features, as a rule, to describe about the wound of entrance, except the appearance at times of bony sand in the track leading to a fractured bone. When a resistant bone has been hit the foyer of fracture will show great loss of substance, the bone will have been very finely comminuted, the pulverized bone will have been driven not only in the direction in which the projectile was travelling but in all directions; and the pulpification of the soft parts will not only be limited to the track of the bullet, but the utter destruction is noticed extending

some distance into the tissues. The wound of exit appears like a bursting forth of the skin; the track leading to the bone is funnel-shaped, the base corresponding to the wound of exit in the skin and the apex to the seat of fracture.

The degree of explosive effects corresponds to the velocity of the projectile at the moment of impact and the resistance offered by the part hit.

The bony structures are not alone in showing explosive effects with high velocities. Dr. Johann Habart, the German military surgeon already referred to, found explosive effects with the projectile of the new Mauser, up to 500 m., in "very vascular tissues, cavities filled with liquid or semi-liquid or viscous masses, such as the skull, heart, liver, spleen, kidneys, stomach, intestines, and bladder, which have been attributed by some observers to hydraulic pressure."

In order to test the influence of hydraulic pressure in causing explosive effects, some interesting experiments were conducted at Frankford Arsenal:

1. Empty powder-cans were fired into at various ranges. The orifices of entrance and exit were found proportional to the size of the projectiles employed. The cans were not deformed and showed no explosive effects.

2. A half-dozen or more powder-cans from the same lot were filled with wet sawdust. The cans were fired into at various ranges. The orifice of entrance in each case presented no special features. The orifice of exit, however, for both projectiles was marked by a bursting forth of the tin and loss of the contents. The cans had expanded as if by an internal force which had been exerted in all directions. The explosive effects were about the same for both bullets.

3. Another lot of powder-cans of the same size and dimensions were filled with water and fired into. The results were quite similar to those found with the wet sawdust, only more extensive, and they were about equal for the two projectiles.

The experience gained by shooting into cadavers at Frankford Arsenal confirms the observations of other investigators

in that explosive effects were *nil* when muscular tissue alone was traversed by either bullet.

The following results were noted with the two projectiles at similar ranges, but beyond 350 yards.

It was necessary to go beyond the latter range to avoid explosive effects, which, as already stated, are often noticed with the projectile of the Experimental Springfield rifle at this range.

Soft Parts.—The wound of entrance corresponds in diameter, as a rule, to the diameter of the projectile. In the middle and remote ranges the entrance wound measured less at times than the diameter of the projectile, but the difference was only apparent, since the wound invariably admitted a projectile of like calibre to the one having inflicted it. In the short ranges it was often noted that skin overlying bone and resistant aponeurosis was apt to show a wound of entrance actually exceeding in diameter that of the missile. The edges of the wound of entrance were at times clear-cut, but more often they were rolled in, and oftened blackened for a distance of a line about the circumference. The latter circumstance gave rise to the notion formerly that the discoloration was due to burning, but from experiments conducted by Beck, also by myself, to be cited later, this fallacy has been forever set at rest.

The wound of exit of the 0.30 calibre rifle was generally larger than the wound of entrance, generally round, marked at times by a mere slit; again it was star-shaped, T-shaped, semicircular, etc.; the edges were usually turned out. When a wound of exit exceeded in diameter that of the projectile to any extent, the circumstance was generally regarded as indicative of bone lesion.

Effects upon Diaphyses of Long Bones.—Up to 350 yards the destructive effects of the two projectiles are alike severe. Unless guided by the wound of entrance or other circumstances it is difficult within this range to determine by the appearance of the injury alone which of the projectiles may have caused it. After this range the destructive effects of the smaller projectile become less than those of the larger missile. The fissuring is

less, the spiculæ of bone are larger, and they are more apt to be attached to the periosteum. These differences are especially noticeable from the 500 to the 1500 yards ranges. At 2000 yards the small bullet again shows rather extensive comminution. This fact has been noted by all observers, and it has been variously explained, though not in a very satisfactory manner. It has been said that the projectile has lost so much of its velocity of translation when it reaches this part of its course that it is apt to lodge, and that the velocity of rotation causes such a disturbance when it is about to engage, that comminution is the result. The angle of impact, which is rarely perpendicular at this range, has been brought forth as a possible cause. Certain it is that a number of the projectiles were observed at this range by us to impinge "side on" at the moment of impact. Whether this circumstance is more apt to occur with reduced charges than we would expect to find in the natural condition is a mooted question.

Delorme and Chavasse state that the Lebel rifle bullet appeared to produce more comminution beyond 1200 m. than the projectile of the Gras rifle. Our experience at Frankford Arsenal does not accord with theirs in this particular, and for very good reason. They experimented with the projectile of the Lebel, which for all practical purposes corresponds to the projectile of our 0.30 calibre Experimental Springfield rifle, and they compared its effects with that of the Gras. The latter is their 11 mm. gun, which in calibre is a trifle less—0.43—than our 0.45 calibre Springfield rifle. This alone would make no great difference, but when we compare the weight of their large calibre with ours we find that our service bullet is 125 grains heavier. This in itself is sufficient to account for the greater destructive effects of our larger calibre projectile over theirs, hence the difference in the results referred to.

Effects upon Joints.—Before engaging upon this part of the subject I wish to preface my remarks by stating that the humane wound of the small-bore gun is especially observed in the joints and soft parts. Owing to the reduction in calibre, the wounds in the latter partake more of the nature of subcu-

taneous wounds, and experience shows that they heal very kindly under appropriate treatment.

It is not necessary in this instance to dwell especially upon the destructive effects of the larger leaden projectile upon joints. Suffice it to say that the greater frontage, which it naturally possesses, is made greater still by deformation, and that these facts, with velocity sufficient to penetrate a joint, serve to convert the 0.45 calibre projectile into a very destructive missile. The following cases noted at Frankford Arsenal will explain the marked difference in destructive effects by the two projectiles upon the spongy ends of bones:

Case I.—Gunshot injury of the lower part of the left femur; bullet marked No. 4; calibre, 0.45; impressed by the velocity common at 100 yards; soft parts frozen. The wound of entrance is marked by a V-shaped tear, each side of the V being 2.5 inches in length; the wound of exit is round, 1.5 inches in diameter. It is funnel-shaped, the base of the funnel corresponding to the skin, and the apex to the point of destruction in the bone. The soft parts adjacent to the track of the bullet are a pulpified mass mixed with finely pulverized bone. One and one-half inches of the external half of the shaft immediately above the articular surface have been carried away; the remaining part of the shaft is separated from the articular surface by a transverse fissure, whilst an oblique fissure extends into the shaft above, 2.5 inches in length. The upper and external quadrant of the patella was chipped. There was no fissuring into the joint. The projectile was not recovered.

Case II.—Gunshot injury of the lower end of the right femur; bullet marked No. 3; calibre, 0.30; impressed by the velocity possessed at 100 yards. The soft parts were frozen, on account of which the explosive effects were enormous. The projectile entered the femur just above the epiphysial junction of the internal condyle and emerged posteriorly in a horizontal line, making an orifice the diameter of the projectile upon entering and five lines in diameter upon leaving the bone. The perforation is well marked. The track of the bullet in the bone and between the latter and the wound of exit is lined

by bony sand. There is a fissure scarcely visible to the naked eye, running downward and from before backward through the articular surface, separating the two condyles almost equally. There is also a subperiosteal fissure, 1.5 inches long, running upward from the point of emergence in the bone; there are a few small splinters of bone near the wound of exit still attached to the periosteum. The projectile was not deformed.

That we should find a perforation so well marked in the spongy end of a bone within the range of explosive effects makes the latter case very remarkable. As it is the only recorded instance of a perforation by the small metallic-jacketed bullet within the explosive zone it is but natural to infer that the frozen condition of the soft parts contributed to the result.

The two following injuries illustrate the difference in destructive effects upon the spongy ends of bones entering into the formation of the knee-joint, at 350 yards.

Case IV.—Gunshot injury of the left tibia and fibula near the knee-joint; bullet marked No. 5; calibre, 0.30; impressed by the velocity possessed at 350 yards. The wound of entrance is round, of the diameter of the projectile; the wound of exit is marked by a longitudinal slit measuring one-third of an inch. The projectile entered the anterior portion of the head of the tibia, one-third of an inch from the articular surface, in the middle line; passing obliquely toward the outer side it emerged from the posterior surface of the head of the fibula. The orifice of entrance in the bone is equivalent in diameter to the diameter of the projectile; the orifice of exit is irregular, and one-third of an inch in its greatest diameter. There is no comminution. The knee-joint was not perforated, but the articular surface adjacent to the tuberosity shows a fissure which was not apparent in the recent state. The projectile was lost.

Case V.—Gunshot injury of the lower shaft of the right femur; bullet marked No. 3; calibre, 0.45; impressed by the velocity common at 350 yards. The wound of entrance is 1.5 inches above the outer condyle, round, and equals in diameter

the diameter of the projectile. The wound of exit is in the upper part of the popliteal space, irregularly quadrilateral in shape, three-fourths of an inch in its greatest diameter. The bullet entered the lower shaft, 1.5 inches above the margin of the articular surface, one-third of an inch outside the median line. Nine large splinters are detached measuring from 3 to 5 inches in length. There are a number of deep vertical fissures in the upper and lower fragments; those in the latter do not invade the joint. The projectile was very much set up.

The tendency on the part of the smaller jacketed bullet to perforate the spongy ends of bones increases as the velocity diminishes. After the 500-yards range, perforations of these structures are rather the rule than the exception. The examples in the specimens before you are sufficiently numerous to substantiate this statement, and they explain themselves so clearly that a recital of each case is not deemed necessary.

We made no notes of injury to bloodvessels. Habart, who mentions some observations in this line, states, with reference to the effects of the 0.30 calibre on the vessels, that "the bloodvessels are seldom torn, and that they are not closed so easily by coagulation as those severed by leaden projectiles. The latter are more apt to lacerate and bruise the bloodvessels, facilitating thereby the formation of thrombi.

"On account of the small aperture in the skin and soft parts in the wounds of the jacketed projectiles, the wounds bleed generally less than those made by the soft leaden bullets; the hemorrhage is easily stopped by coagulation, which forms a spontaneous cure under a blood scab."

Deformation of Projectiles.—The deformations of projectiles which accompany the majority of the specimens represent nearly every form of alteration known. Those of the leaden projectiles are sufficiently familiar and require no comment. The deformation of the jacketed projectile is most common at the conical end, consisting usually in a slight dent or flattening or partial separation of the metallic mantle from the leaden nucleus; complete separation between the jacket and nucleus is a very rare occurrence. It occurs principally

with high velocities at close range, when the projectile encounters resistant bone.

The Heat Imparted to Projectiles.—The heat imparted to a projectile by the ignition of the powder, the resistance in the barrel, etc., has been very much exaggerated. Some have gone so far as to claim that the small jacketed bullets are rendered aseptic thereby. In a series of experiments conducted at the Pathological Laboratory of the Johns Hopkins University and Hospital, also at Frankford Arsenal, I was able to show that this claim is false. Animals shot with jacketed projectiles previously infected with anthrax bacilli almost invariably died of anthrax. In one instance erysipelas was communicated to a rabbit by shooting him with a 0.45 calibre leaden projectile from a Colt's revolver, the projectile having been previously infected with the streptococcus of erysipelas.

DISCUSSION.

Dr. G. G. Davis: There seems to be a false opinion prevalent with regard to the destructiveness of these rifles. I believe that the object of the Government was not to produce a weapon that would inflict a more severe injury, but simply to reduce the trajectory and lengthen the range, thereby increasing the liability of wounding. That could only be done by having great initial velocity and lessening the resistance; this necessitated modifying the size of the ball. During the last thirty years projectiles have undergone marked changes. There was first the leaden round or conical ball, next the hardened conical bullet, and now the small bullet with hardened jacket. The round ball makes a different wound from the conical; it does not crush, nor tear, nor exhibit the so-called explosive effect to the extent which the conical ball does. This is when both are of the same calibre. The injury done by the bullet shown to-night is less than that done by the ordinary service 0.45 calibre. This is the case to such an extent that hunters who aim to get immediate results discard small calibres. For that reason, as a means of disabling a person, the 0.30 calibre ball does not compare favorably with the 0.45 calibre.

It has been said that the explosive effects were the same for both bullets up to three hundred and fifty yards. This may be so, but I do not see how it can be. I believe the specimens fail to show it. The injury done by the

0.45 calibre is greater than that done by the 0.30 calibre. The explosive effects are directly proportional to the velocity, and inasmuch as the velocity of the new ball is greater than that of the large ball, the explosive effects should be greater. If the balls were of the same size, I believe that they would be greater, but the 0.45 calibre ball has a greater surface of impact, which may counterbalance the increased velocity of the smaller ball. The specimens seem to be at variance with the experiments in regard to the cans. As to the crushing effects at twelve hundred yards I do not think that we can draw any inference, as the statements of the paper on that point seem to be at variance, and the bullets do not seem to always strike point-on.

As regards the effect which this will have practically on the wounds produced, the actual injury is certainly less in almost all cases with the 30-calibre ball. In a gunshot wound we have not only the direct injury to the part, but we also have sepsis conveyed to the wound. Dr. La Garde has shown that sepsis may be conveyed by the ball itself and by the ball carrying portions of the clothing, etc., into the wound. This danger is much increased by the larger size of the old bullet.

Another point is the fact that these 0.30 calibre balls with German-silver jackets have been little, if at all, deformed after having passed through bone. The balls that were used in the War of the Rebellion were almost pure lead, and when they struck bone they spread out, making a much more serious injury. When the 0.45 calibre was adopted, a harder projectile and larger charges were employed. The hardened bullet contains one part of tin to sixteen of lead. In the treatment of the injuries produced by these small bullets the adoption of antiseptic measures will be more valuable than in the wounds produced by the ordinary 0.45 calibre ball. It emphasizes the necessity of the soldier being furnished with an antiseptic packet, so that should he become wounded an antiseptic occlusive dressing can at once be applied, and thus the wound be prevented from becoming further infected.

Dr. John H. Brinton: The prominent feature that strikes me in looking at these specimens is that the injury has been produced for the most part by a ball striking point-foremost. These experimental results have been effected, I understand, by a charge graded down so that a diminished charge at close range represents a great distance—twelve hundred yards, as stated by Dr. Hinsdale. The question naturally arises, whether this series of specimens really represents the injuries we should meet with in the field. I have had the opportunity of examining many cases of gunshot wounds at long range, and have been impressed with the fact that very few of the injuries were produced by projectiles flying directly point-foremost. It is probable that at long range the balls shown to-night would not strike point-on. Their trajectory must be influenced, as in the flight of all other projectiles, by wind currents and gravity. In experimental researches at short range—fifty-three feet, as stated in the report—these influences would be minimized, practically abolished. It must also be remembered that the effects obtained

by firing bullets into dead bodies is not the same as those obtained in living tissues.

In general, the effect of the new, smaller, and jacketed projectiles is apparently more humane than those hitherto employed. It can be summed up in greater velocity, greater penetrating power, lower trajectory, diminished crushing results, and probably less fatal consequences. As ammunition their comparatively slight weight and portability is an advantage.

INDEX.

Abbott, A. C., 122, 242
Abscess, case of liver, 82
Additions to the Mütter Museum, lxxxiii
Agnew, D. Hayes, memoir of, xxix
Anæsthesia, chloroform, 137, 198
Aneurism, case of enormous, 178
Annual address of President, lxxvii

Bleeder, peliosis rheumatica in a, 100
Blood-count, effect of massage on the, 240
Brinton, John H., 257

Cæsarean sections, two, 235
Case of enormous aneurism, 178
Chloroform anæsthesia, 137, 198
Chromidrosis, red, 133
Chronic valvular disease of the heart, 89
Cleft palate, both hard and soft, and hare-lip, 181
Cohen, J. Solis-, 191, 197
Cohen, S. Solis-, 215, 218, 221, 222
Correction of inveterate pes varus, 225

Davis, E. P., 50
Davis, G. G., 213, 225, 256
Dercum, Francis X., 48
De Schweinitz, E. A., 110
De Schweinitz, G. E., 119
Dulles, Charles W., 197
Dunn, Thomas D., 100, 102, 103, 109

Einhorn's gastrodiaphane, 222
Etiology of membranous rhinitis, 122
Evans, H. Y., 213

Forbes, W. S., 181, 196, 217

Gastrodiaphane, Einhorn's, 222
Griffith, J. P. Crozer, 88

Hare, H. A., 98, 137, 176, 177, 179, 206, 212, 214, 216, 241
Hare-lip and cleft palate, 181
Harris, Robert P., 60, 239
Harte, Richard H., 213
Hartzell, M. B., 133
Heart, chronic valvular disease of, 89
Hewson, A., 234
Hinsdale, Guy, 243
Hirst, Barton Cooke, 47, 62, 235

Inveterate pes varus, correction of, 225

Kidney, stone in, 1

La Garde, Louis A., remarks upon the comparative difference between certain wounds inflicted by the projectiles of the large and small calibre hand-weapons, 243
Larynx, voice without the, 191
Leidy, Joseph, Jr., 242
Leukæmia, with rare lymphoid growths of orbits and parotid glands, 103
Life and character of George Cheyne Shattuck, lxvi
Liver abscess due to disease of the vermiform appendix, 82
Lloyd, James Hendrie, 49, 64, 79

Massage, effect of, on the blood-count, 240
Massey, Isaac, 189
Meigs, Arthur V., 78
Membranous rhinitis, etiology of, 122
Memoir of D. Hayes Agnew, xxix
Mills, Charles K., 17
Mitchell, J. K., 188, 240, 242
Mitchell, S. Weir, lxxvii, 214
Morton, T. S. K., 234
Mütter Museum, additions to the, lxxxiii

Neural affections of puerperal origin, 17
Neuritis and myelitis, and the forms of paralysis and pseudo-paralysis following labor, 17
Noble, Charles P., 51
Norris, William F., 109

Œsophageal stethoscope, with remarks on intra-thoracic auscultation, 218
Ophthalmology, sepsis in, 110
Osler, William, 102

Peliosis rheumatica in a bleeder, 100
Piperazin in treatment of stone in kidney, 1
President, annual address of, lxxvii

Randall, B. Alexander, 242
Red chromidrosis, 133
Reichert, Edward T., 175
Results of a bacteriological examination of the pipettes and collyria taken from a treatment case used in ophthalmic practice; with the effects of inoculation, 110

Sepsis in ophthalmology, 110
Shattuck, George Cheyne, notice of the life and character of, lxvi
Shoemaker, George Erety, 82
Sinkler, Wharton, 48, 80
Stethoscope, œsophageal, 218
Stewart, David D., 1, 221, 223
Stillé, Alfred, lxvi
Stone in kidney, 1
Study of the influence of chloroform on the respiration and circulation, 137
Symphysiotomies, three, 235
Symphysiotomy *versus* its substitutes; with the report of a case of symphysiotomy, 51
Synchronous amputation of both thighs for railroad injury, 189
Syringomyelia, with exhibition of sections of the spinal cord, 64, 80

Taylor, J. Madison, 217
Thighs, amputation of both, 189
Thornton, E. Q., 137
Tyson, James, 89, 99, 180

Voice without the larynx, 191

White, J. William, xxix
Wood, H. C., 174, 177, 198, 211, 212, 215, 217
Woodbury, Frank, 175, 215, 217 223